Twenty-fifth Annual Meeting
Atlantic City, June 3-7, 1959

VOLUME XXXIV NUMBER 6

DISEASES

of the

CHEST

OFFICIAL PUBLICATION

AMERICAN COLLEGE CHEST PHYSICIANS

PUBLISHED MONTHLY

DECEMBER
1958

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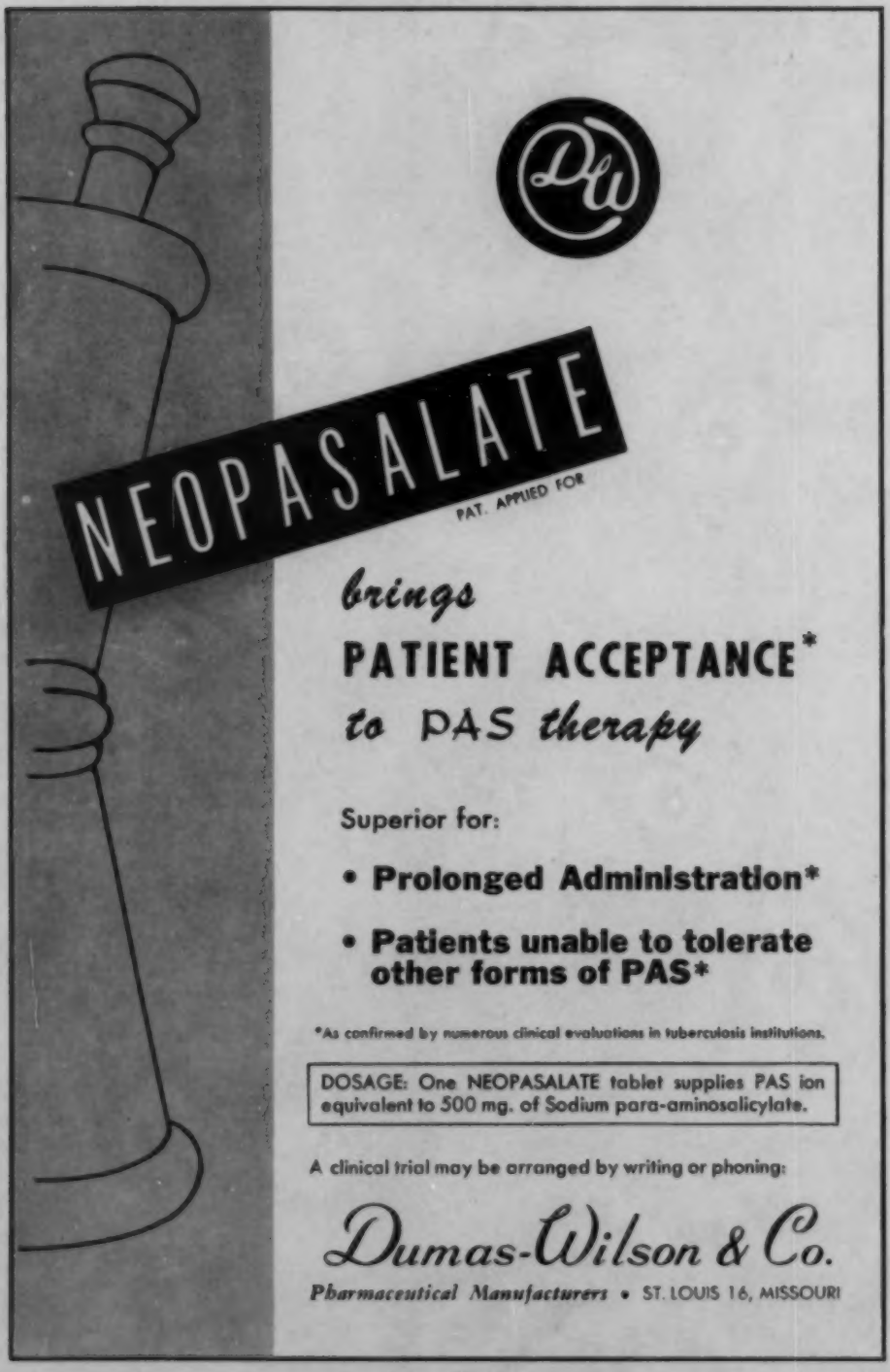
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DISEASES *of the* CHEST

OFFICIAL PUBLICATION

OF THE

AMERICAN COLLEGE OF CHEST PHYSICIANS

EXECUTIVE OFFICE

112 East Chestnut Street, Chicago 11, Illinois, U.S.A.

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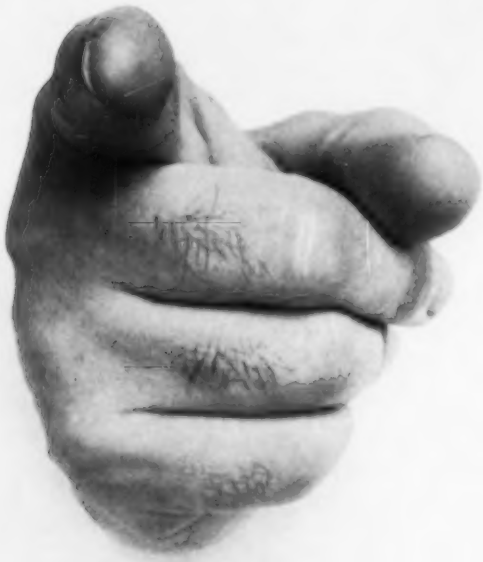

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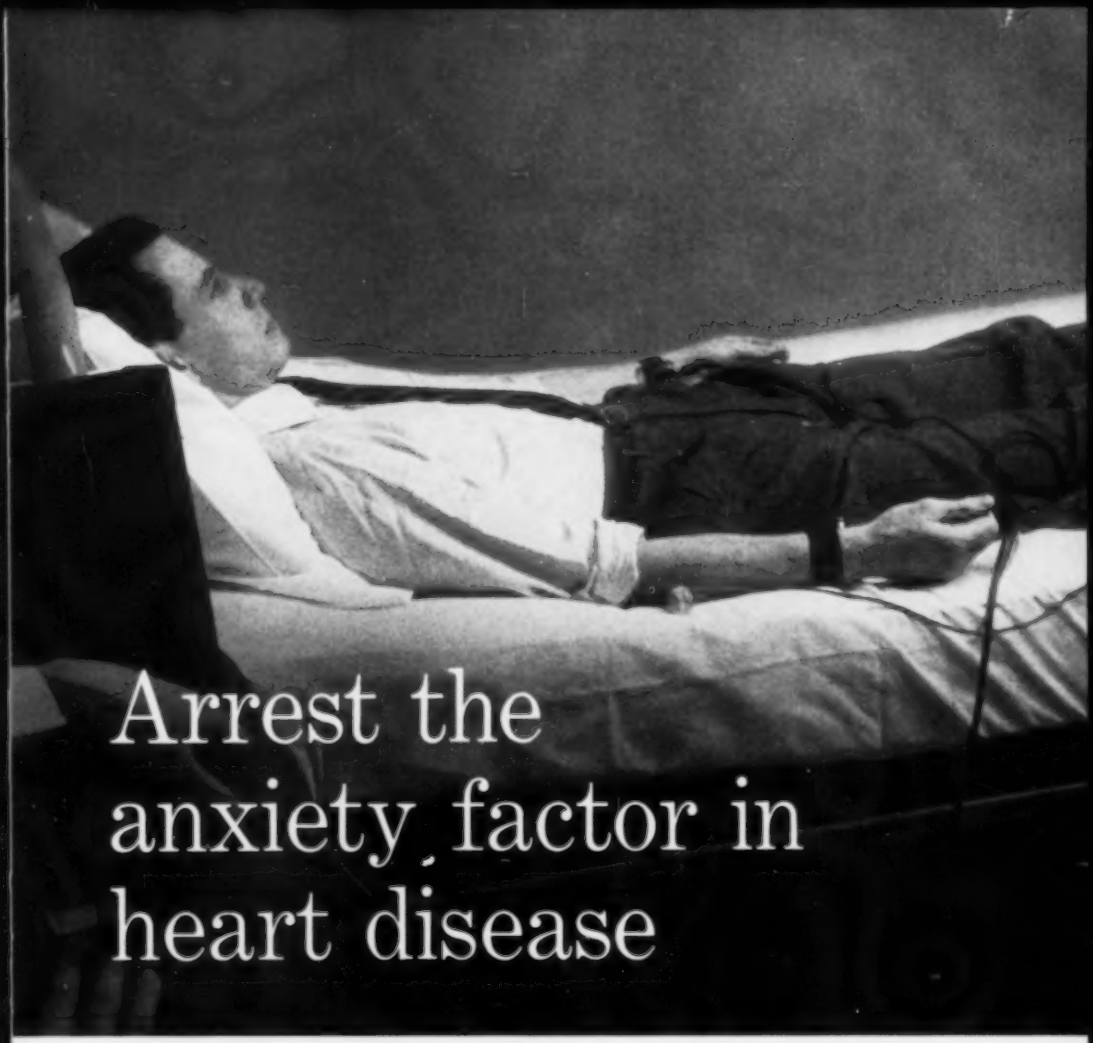

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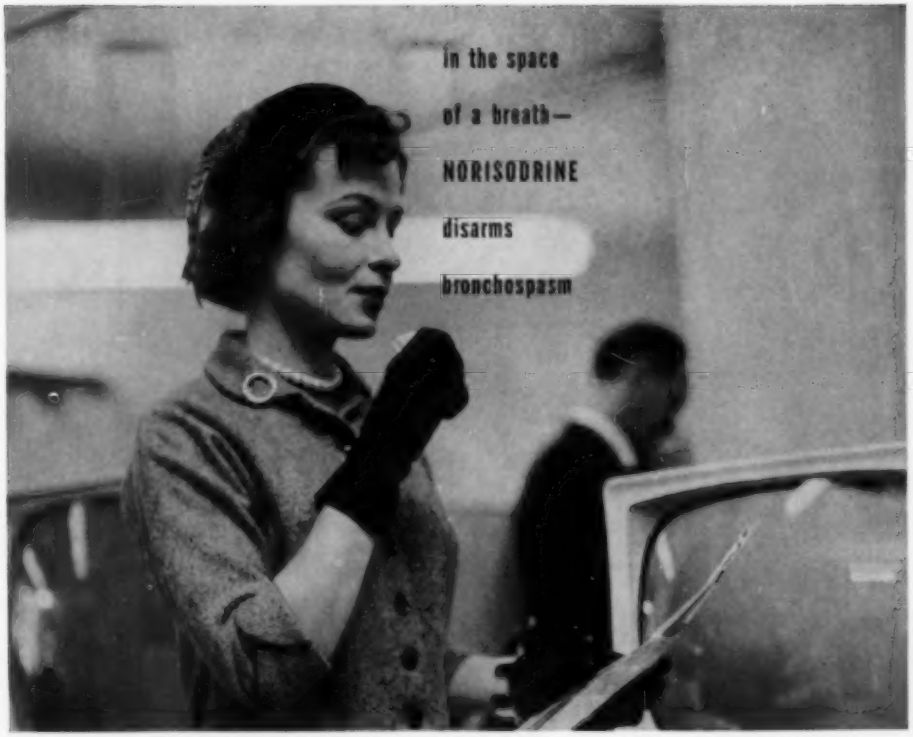

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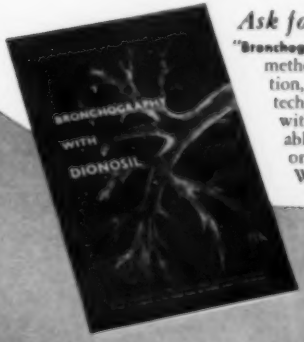

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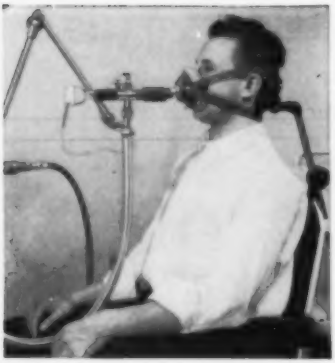

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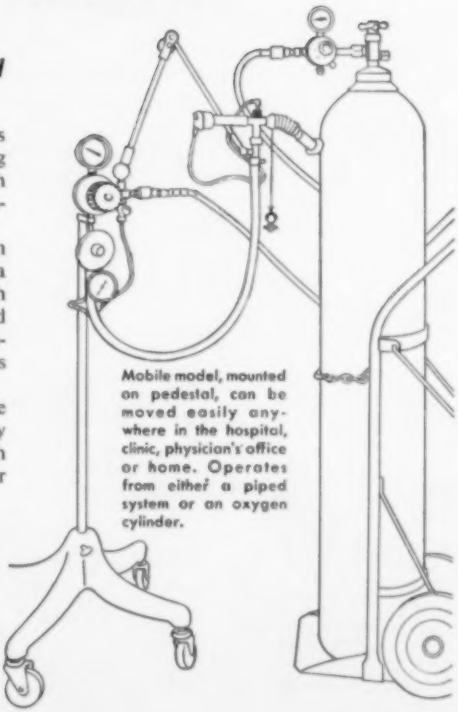

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a rapidly eliminated bronchographic medium...

less irritating—rarely penetrates the alveoli

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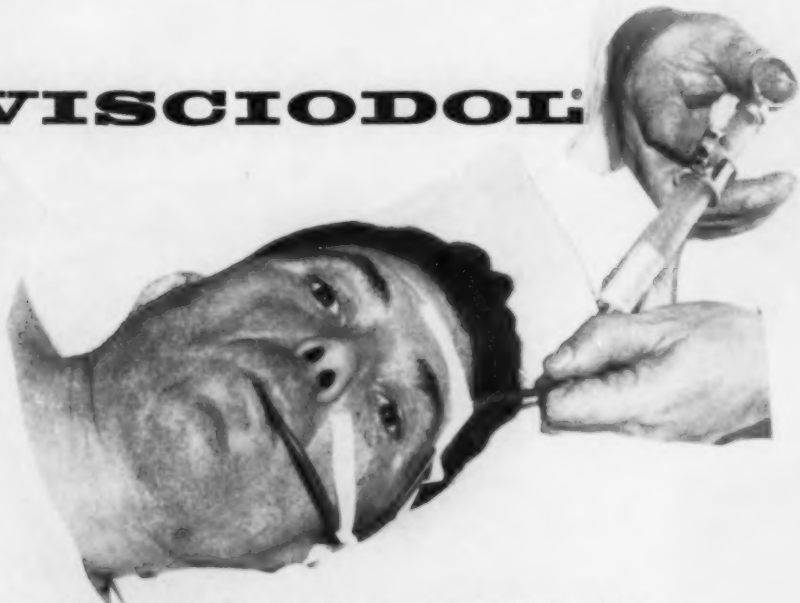

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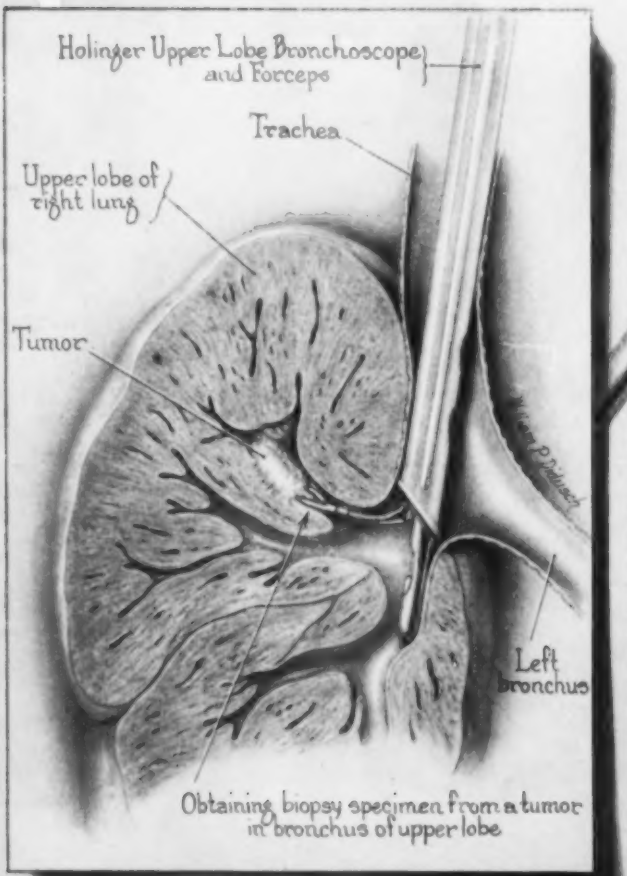
Cat. No. 4930



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The bronchoscopic tube is 8 mm. in diameter and 40 cm. long with oxygen inlet. The tube is distally lighted, and the proximal end is funnel-shaped. The operating telescope contains a right angle system for visualization of the upper lobe bronchi. A deflecting mechanism actuated by two knurled knobs at the proximal end of the telescope directs the biopsy forceps, aspirator, or other device under the telescopic vision in the direction required. A channel is provided in the tube for inserting various instruments.

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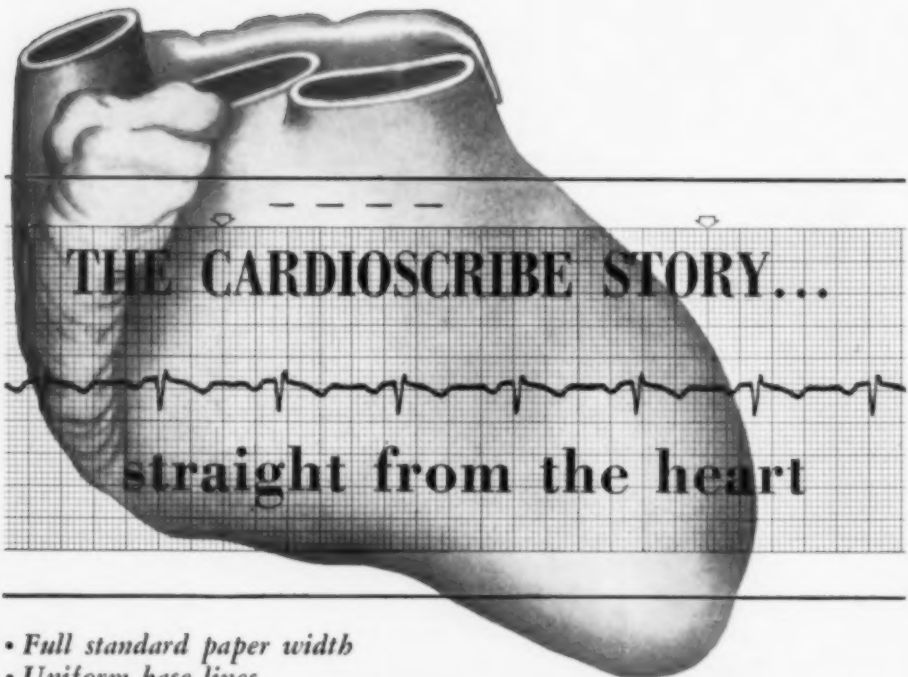

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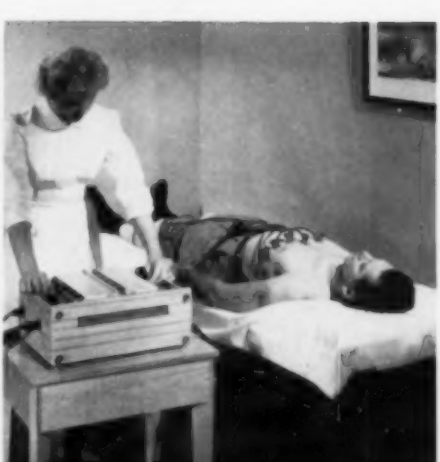

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When anxiety and tension complicate **HIGH BLOOD PRESSURE**

Treatment is more successful when mental pressure and blood pressure are treated together

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Miltown in addition to ganglionic blocking therapy resulted in subjective and objective improvement in 35 of 37 patients. On the antihypertensive agent alone, only 28 patients improved.[1]

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1. Nussbaum, H. E., Leff, W. A., Mattia, V. D., Jr. and Hillman, E.: An effective combination in the treatment of the hypertensive patient. Am. J. M. Sc. **234**: 150, Aug. 1957.

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THE COMPLETE Rx FOR COUGH CONTROL

cough sedative / antihistamine / expectorant

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Each teaspoonful (5 cc.) of HYCOMINE contains:

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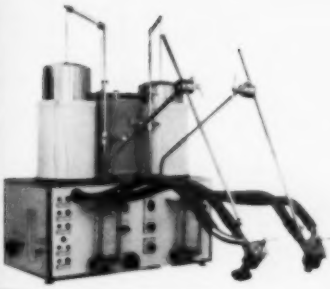

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(black and white)
250 mg., 125 mg.
(for pediatric or long-term therapy)

ORAL SUSPENSION
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For relief of symptoms and malaise of the common cold and prevention of secondary complications

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REFERENCES: 1. Carlozzi, M.: Antibiotic Med. & Clin. Therapy 5:146 (Feb.) 1958. 2. Welch, H.; Wright, W. W., and Staffa, A. W.: Antibiotic Med. & Clin. Therapy 5:52 (Jan.) 1958. 3. Marlow, A. A., and Bartlett, G. R.: Glucosamine and leukemia, Proc. Soc. Exp. Biol. & Med. 84:41, 1953. 4. Shalowitz, M.: Clin. Rev. 1:25 (April) 1958. 5. Nathan, L. A.: Arch. Pediat. 75:251 (June) 1958. 6. Cornbleet, T.; Chesrow, E., and Barsky, S.: Antibiotic Med. & Clin. Therapy 5:328 (May) 1958. 7. Stone, M. L.; Sedlis, A., Bamford, J., and Bradley, W.: Antibiotic Med. & Clin. Therapy 5:322 (May) 1958. 8. Harris, H.: Clin. Rev. 1:15 (July) 1958.

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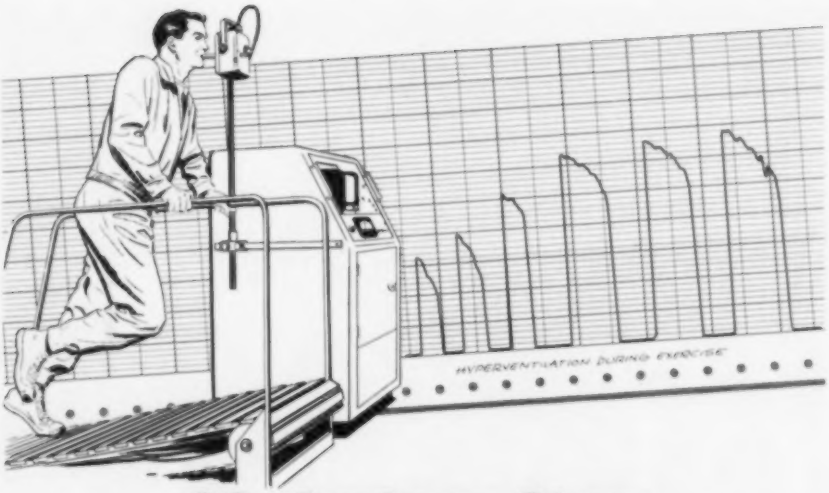

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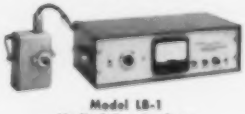

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to treat more patients more effectively

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Virtual **absence of diabetogenic activity, edema, sodium or water retention, hypertension, or psychic reactions** has been noted with DECADRON.[1,2,3,4] Other "classical" reactions were less frequent and less severe. DECADRON showed no increase in ulcerogenic potential, and **digestive complaints were rare. Nor have there been any new or "peculiar" side effects,** such as muscle wasting, leg cramps, weakness, depression, anorexia, weight loss, headache, dizziness, tachycardia, or erythema. Thus DECADRON introduces **a new order of magnitude in safety,** unprecedented in corticosteroid therapy.

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More patients can be treated **more effectively** with DECADRON. Its higher anti-inflammatory potency frequently brings relief to cases resistant to other steroids. Virtual freedom from diabetogenic effect in therapeutic dosage permits treatment of many diabetics without an increase in insulin requirements. Absence of hypertension and of sodium and fluid retention allows effective therapy of many patients with cardiovascular disorders. Reduction in the incidence and severity of many side effects extends the benefits of therapy to numerous patients who could not tolerate other steroids. And a healthy sense of well-being, reported by nearly all patients on DECADRON, assures greater patient cooperation.

References:
1. Boland, E. W.: California Med. 88:417 (June) 1958. 2. Bunim, J. J., et al.: Arthr. & Rheum. 1:313 (Aug.) 1958 3. Boland, E. W., and Headley, N. E.: Paper read before the Am. Rheum. Assoc., June 21, 1958, San Francisco, Calif. 4. Bunim, J. J., et al.: Paper read before the Am. Rheum. Assoc., June 21, 1958, San Francisco, Calif.

To treat more patients more effectively

in all allergic and inflammatory disorders

amenable to corticosteroid therapy

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one 4 mg. tablet of	one 5 mg. tablet of	one 20 mg. tablet of	one 25 mg. tablet of
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SUPPLIED:
As 0.75 mg. scored pentagon-shaped tablets; also as 0.5 mg tablets, to provide maximal individualized flexibility of dosage adjustment.

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Detailed literature on DECADRON is available to physicians on request.

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The great corticosteroid era opened ten years ago with the introduction of Cortone® (Cortisone). Today, MERCK SHARP & DOHME proudly presents the crowning achievement of the first corticosteroid decade—DECADRON (dexamethasone)—a new and unique compound, which brings a new order of magnitude to corticosteroid therapy.

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DEXAMETHASONE

to treat more patients more effectively

MSD MERCK SHARP & DOHME

DISEASES of the CHEST

VOLUME XXXIV DECEMBER, 1958 NUMBER 6

Status of Federal-State Cooperative Tuberculosis Eradication

A. F. RANNEY, D.V.M.

Washington, D. C.

Remember the World Series game a few years back, when the Yankees were ahead one to nothing and had a no hitter going for them? Then in the last half of the ninth, with two men out, Cookie Lavagetto hit a double that won the game for the Dodgers. That was something to see and be a part of, and I've always felt a little sorry for the spectators who got up and left at the start of that last half-inning thinking the game was all over.

You know, it's bad enough to see the spectators leave a game before the last man is out, but when the players themselves start leaving—well, then it's time for somebody to take another look at the game.

That is exactly what I suggest we do today with the tuberculosis eradication program. The fight against bovine tuberculosis is not over until the last source of infection is wiped out—but some of the players have already started to leave the field. So, it's time to take another look at the whole idea of the game.

That idea is to see tuberculosis eradicated, not quit because we think we have a comfortable lead.

In the last few years, the reports to this Association on the status of our cooperative tuberculosis eradication program have consistently called attention to the progress we've made in reducing the incidence of the disease to its present low level. Now this makes excellent historical background—fine statistics on runs batted in and so forth. But the past is past, and reports of past progress are of little help to us in actually finishing the job—especially if they impress us so much that we become complacent and satisfied with what we've already done.

The plain truth is that bovine tuberculosis is still with us, and we haven't won the fight or done our job till we've mopped up the last traces of it.

Our past victories should serve simply to show us that we can win the final round. We *can* eliminate tuberculosis. We have but to revitalize our efforts, "dig in and stamp it out," rather than rest on the laurels of past accomplishments and allow control measures to be substituted for eradication.

*Read before the 34th annual meeting of the United States Livestock Sanitary Association, St. Louis Missouri, November 15, 1957.

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It is unfortunate, indeed, that severe outbreaks that practically eliminate some of our valuable herds seem inevitable before we are awakened to the necessity for more concentrated action.

It is high time that this organization, the livestock industry, and the veterinary profession cast aside the prevailing lackadaisical attitude toward

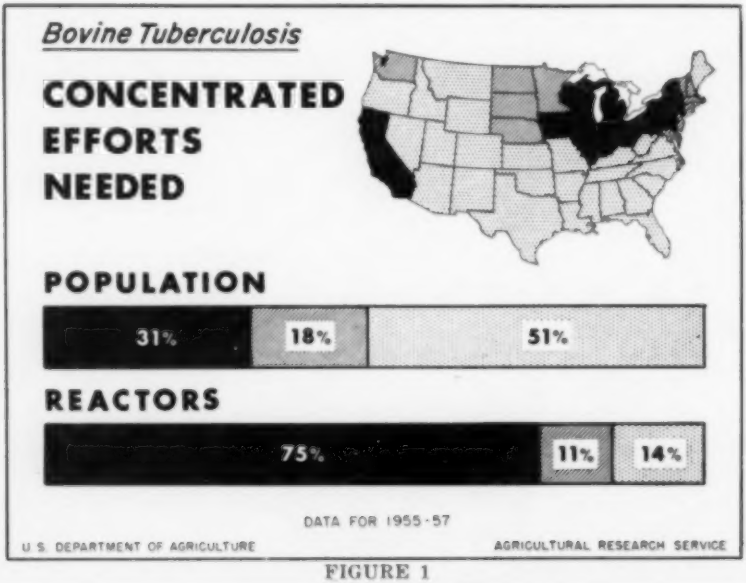

FIGURE 1

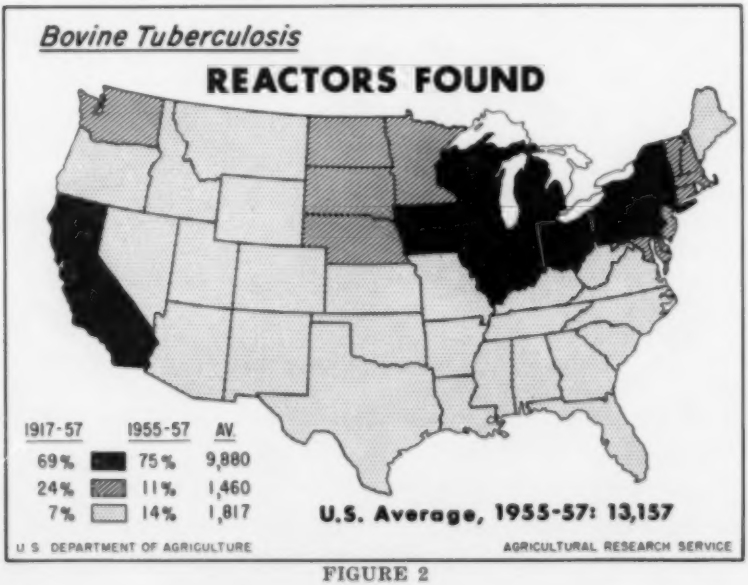

FIGURE 2

this disease and take another look at the goal of our cooperative project: tuberculosis *eradication*. And take a look, too, at some of the things that are delaying the attainment of that goal.

Figure 1 shows where we have found the majority of the tuberculosis reactors since the eradication program began 40 years ago. Of the more than four million reactors found so far, 69 per cent have come from nine States and 24 per cent from 13 other States—which makes a total of 93 per cent from 22 States, while only 7 per cent of the reactors came from the remaining group of 26 States.

The same chart carries the figures on location of reactors for the past three years. In the same nine States, we've found 75 per cent of the total reactors in the United States; an additional 11 per cent came from the 13 States—making a total of 86 per cent of recent tuberculosis reactors in the same 22 States, and only 14 per cent from the remaining 26 States.

Figure 2 shows the total cattle population in the various areas. In the nine-State area where 75 per cent of the country's reactors were found in the past three years, only 31 per cent of our national cattle population is located. The 13 States where 11 per cent of the reactors were found have 18 per cent of all our cattle, and in the other 26 States where only 14 per cent of the reactors were discovered, you will find over half of the cattle in the country.

A careful review of the funds available for tuberculosis eradication suggests that in several States where relatively few reactors are being found, Federal funds will of necessity be reduced in order to support a more intensified program in other States, where a larger number of infected animals are presently being detected.

That's one new shot-in-the-arm we hope to give the tuberculosis eradication project. Then we have the fundamental procedures for disease eradication, and if these procedures are going unused as far as tuberculosis is concerned, it's time to dust them off and put them to work. I have these five points that must be given special attention:

1. First, we must reaffirm our confidence in the tuberculin test, which when properly applied and interpreted is recognized as one of the most dependable disease detection tests we have. All too frequently, undue concern is expressed because some animals that react to the tuberculin test do not show up with gross lesions of tuberculosis on post mortem inspection. The best scientific evidence and observations of millions of carcasses clearly show that the tuberculin test is more reliable than the customary post mortem examination as a means of detecting tuberculosis.

During the past year, a set of 60 color slides was distributed to each of our Animal Disease Eradication Division field offices and veterinary colleges. These slides were developed for use in reacquainting veterinarians and acquainting veterinary students with the proper technique and procedures for applying and interpreting tuberculin tests. The slides also provide additional educational information. A series of leaflets, "TB Topics," is also being distributed through the field stations to veterinarians

who may be called upon to apply tuberculin tests. We hope these aids and others to be developed will help in redirecting attention to the approved uniform testing procedures.

2. My second point is this: We must give close attention to the proper quarantine of infected herds, and make certain that no known-infected or known-exposed animal is the cause of spreading the disease.

You can see how important it is to give careful attention to quarantined herds when you realize that only about 4 per cent of the total tests during a year are made in quarantined herds, and yet that's where we find more than one-fourth of all our reactors.

3. My third point: You can't get along without *C* and *D* in tuberculosis. We must assure ourselves that all premises that have harbored tuberculous animals are properly *Cleaned* and *Disinfected* under vigilant supervision.

4. The fourth item I'll mention concerns an area that needs some real tightening up. I guess in baseball lingo you'd call it "scouting" but we know it as epizootiological investigation. We must work at this carefully, thoroughly, and vigorously. We have had tremendous success in locating many centers of infection as a result of tracing procedures, and so it is especially difficult to understand the seeming lack of interest by some individuals in this phase of the project. They apparently take for granted that periodic testing alone is sufficient. But neglect in making every reasonable effort to trace the origin of known-infected animals, or to determine the health status of herds that harbor animals previously exposed in other herds, *just cannot be justified.*

We must trace to their herds of origin all animals that show lesions of tuberculosis on regular kill. We must determine the origin of animals that react to tuberculin tests, and we must follow-up on exposed animals removed from infected herds. In each case we must apply the appropriate tests to the herds that contain, or that may have harbored, known-tuberculous animals or those exposed to infection.

These tracing procedures have had relatively little attention in connection with tuberculosis eradication until recent years, but they are proving extremely valuable in locating infected herds that might not be detected until an area test may be applied some time in the future.

Our tracing procedures sorely need a system that will provide for adequate identification of individual animals and complete records covering their movements. Such information would be most helpful in our work, and we hope that more consideration will be given to this aspect of the program.

A little while ago, I said that our epizootiological investigations must be thorough, and they must be. They must be carried through to include possible association with humans, poultry, and classes of livestock other than cattle that may be infected with tuberculosis of any type.

During the past year, a relatively large number of bovine carcasses inspected on regular kill in one slaughter plant were reported with tuberculous lesions. About 70 per cent of these reports have shown that only

mesenteric lesions were found. Investigations as to the origin of the animals that had these lesions, as well as limited laboratory studies, suggest that tuberculosis of the avian type may be associated with some of the cases. Complete laboratory tests have not yet been made to determine conclusively the type (or types) of tuberculosis that may be involved.

We anticipate better laboratory facilities in the near future to help in our diagnostic and investigational work on tuberculosis. These new facilities will be a tremendous asset to the overall program.

It is generally agreed that more effort should be directed toward the eradication of tuberculosis in poultry and swine—to help reduce to some degree tuberculosis in cattle. Federal meat inspection records show that 3 per cent of swine slaughtered are retained due to tuberculosis. If slaughter swine could be adequately identified so that those showing lesions could routinely be identified with the farms of origin, we could accomplish a great deal with a minimum of effort and expense in locating tuberculosis in cattle, swine, and poultry on these farms. Possibilities of obtaining help through the use of a tattoo system, presently in use to identify certain lots of market swine, are being investigated.

5. And then, finally, we must put forth the kind of effort in our tuberculosis eradication work that will gain the most with the least expenditure of funds. We must give careful consideration to how often complete area tests are necessary in certain localities, and we must be judicious in selecting herds in other localities where we may be able to achieve eradication without periodic testing of all cattle. I have already mentioned the redistribution of funds to pursue a more concentrated program in those States where most of the infected animals are now being found.

You might call all of this the "coach's pep talk" the "give-em-heck-and-go-out-there-and-win" approach. But let me say this in closing—a coach may have a good team, but he must consider the possibility of losing. We may have a low incidence of bovine tuberculosis in this country, but all of us familiar with the program are constantly faced with the fact that many dangerous reservoirs of infection exist to menace our livestock. We've been sliding along with a low incidence for a good many years. Now is the time to revitalize our efforts and actually eradicate tuberculosis.

SUMMARY

1. The progress that has been made in bovine tuberculosis eradication has lead to complacency and a general feeling that the disease has been eradicated. We should revitalize our efforts to eradicate tuberculosis. Our past victories should serve to show that we can reach our goal of complete eradication.

2. A majority (69 per cent) of the tuberculosis reactors found during the period 1917-1957 inclusive came from nine States. During the past three years (1955-1957 inclusive) the same nine States yielded 75 per cent of the total reactors.

3. Five fundamental points must be given special attention. They are:

1. Reaffirm our confidence in the tuberculin test, which when properly applied and interpreted is recognized as one of the most dependable disease detection tests we have. All too frequently, undue concern is expressed because some animals that react to the tuberculin test do not show up with gross lesions of tuberculosis on post-mortem inspection. The best scientific evidence and observations of millions of carcasses clearly show that the tuberculin test is more reliable than the customary post-mortem examination as a means of detecting tuberculosis.

2. We must give close attention to the proper quarantine of infected herds, and make certain that no known infected or known-exposed animal is the cause of spreading the disease.

3. We must assure ourselves that all premises that have harbored tuberculous animals are properly cleaned and disinfected under vigilant supervision.

4. We have had tremendous success in locating many centers of infection as a result of epizootiological investigations. Periodic testing alone is not enough. We must trace to their herds of origin all animals that show lesions of tuberculosis on regular kill. We must determine the origin of animals that react to tuberculin tests, and we must follow-up on exposed animals removed from infected herds. More effort should be directed toward the eradication of tuberculosis in poultry and swine.

5. We must redistribute funds to pursue a more concentrated program in those States where most of the infected animals are now being found.

RESUMEN

1. Los progresos logrados en la erradicación de la tuberculosis bovina, han llevado a la complacencia y a un sentimiento general de que la enfermedad ha sido erradicada. Debemos revitalizar nuestros esfuerzos para erradicar la tuberculosis. Nuestras victorias pasadas deben servir como demostración de que podemos alcanzar nuestra meta de la erradicación completa.

2. La mayoría de los reactores de tuberculosis (69 por ciento) encontrados durante el período de 1917 a 1957 inclusive, vinieron de nueve estados. Durante los pasados tres años (1955-1957 inclusive) los mismos nueve estados rindieron el 75 por ciento del total de reactores.

3. Cinco puntos fundamentales deben tener especial atención:

1. Reafirmar nuestra confianza en la reacción tuberculínica que cuando es bien aplicada e interpretada se reconoce como uno de los procedimientos para descubrir la enfermedad más digno de confianza. Demasiado frecuentemente se ha expresado una preocupación indebida porque algunos animales que reaccionan a la tuberculina no muestran lesiones gacroscópicas de tuberculosis en la necropsia.

La mejor evidencia científica y la observación de millones de cuerpos de animales claramente demuestran que la prueba de la tuberculina es

más de confiar que el examen corriente postmortem como medio de descubrir la tuberculosis.

2. Debemos prestar atención estrecha a la adecuada cuarentena del ganado infectado y cerciorarnos de ningún animal conocido como infectado o que se sabe ha estado expuesto, sea la causa de diseminación de la enfermedad.

3. Debemos asegurarnos de que todos los lugares que han alojado animales tuberculosos sean limpiados adecuadamerte y desinfectados bajo estrecha supervisión.

4. Como resultado de investigaciones epizootiológicas hemos logrado un éxito magnífico para localizar centros de infección. La prueba periódica no basta, debemos seguir la huella de la infección hacia los grupos del ganado de origen cuando encontremos animales infectados en la matanza habitual.

Debemos determinar el origen de los animales que reaccionan a la tuberculina y realizar el seguimiento de los animales que se han desplazado de sus grupos infectantes. Mayores esfuerzos deben hacerse hacia la erradicación de la tuberculosis entre las aves y los porcinos.

5. Debemos hacer nueva distribución de los fondos para proseguir un programa más concentrado en aquellos estados donde se encuentran ahora más animales enfermos.

RESUME

1. Le progrès qui a été fait dans l'éradication de la tuberculose bovine a conduit à penser complaisamment et à imaginer d'une façon générale que la maladie avait été éradiquée. Nous devrions donner une flamme nouvella à nos efforts pour venir à bout de la tuberculose. Nos victoires passées devraient servir à montrer que nous pouvons atteindre notre but d'éradication complète.

2. La majorité (69%) des cas réagissant à la tuberculine découverte pendant la période de 1917 à 1957 inclusivement est venue de neuf Etats. Pendant les trois dernières années (1955, 1957 inclus) les mêmes neuf états donnèrent 75% de la totalité des allergiques.

3. Cinq points fondamentaux devraient retenir spécialement notre attention. Ce sont:

1. Réaffirmer notre confiance dans le test tuberculinique qui, lorsqu'il est convenablement pratiqué et interprété, est reconnu comme l'un des tests de détection les plus défendables que nous ayons. On fait souvent état à tort du fait que certains animaux qui réagissent au test tuberculinique ne montrent pas de lésions volumineuses de tuberculose à l'examen postmortem. La démonstration la plus scientique et les observations de millions de corps d'animaux montrent clairement que le test tuberculinique est plus valable que l'examen autopsique de routine comme moyen de détecter la tuberculose.

2. Nous devons prêter la plus grande attention à faire respecter la

quarantaine des troupeaux infectés, et être certains qu'aucun animal infecté connu ou exposé connu n'est la cause de l'extension de la maladie.

3. Nous devons nous-mêmes nous assurer que toutes les propriétés qui ont abrité des animaux tuberculeux sont convenablement nettoyées et désinfectées sous supervision vigilante.

4. Nous avons eu un énorme succès en localisant beaucoup de centres d'infection à la suite de la recherche des épizooties. Le seul contrôle périodique n'est pas suffisant. Nous devons poursuivre jusqu'à leurs troupeaux d'origine tous les animaux qui montrent des lésions de tuberculose lors de l'abattage. Nous devons déterminer l'origine des animaux qui réagissent aux tests tuberculiniques et nous devons contrôler les animaux exposés sous-traits aux troupeaux infectés. Un plus grand effort devrait être dirigé vers l'éradication de la tuberculose chez la volaille et les porcs.

5. Nous devons redistribuer des fonds pour poursuivre un programme plus important dans les Etats où l'on trouve maintenant la plupart des animaux infectés.

ZUSAMMENFASSUNG

1. Die Fortschritte, die in der Ausmerzung der bovinen Tuberkulose gemacht wurden, haben zu einer Selbstgefälligkeit geführt und zu der allgemeinen Überzeugung, dass die Krankheit ausgelöscht sei. Wir müssen unsere Bemühungen zur Beseitigung der Tuberkulose neu beleben. Unsere zurückliegenden Siege sollten als Hinweis darauf nützlich sein, dass wir unser Ziel der vollständigen Ausmerzung erreichen können.

2. Eine Mehrheit (69%) von während der Zeitspanne von 1917 bis einschliesslich 1957 gefundenen Personen mit positiver Tuberkulin-Reaktion kamen aus 9 Bundesländern. Während der letzten 3 Jahre (1955 bis einschliesslich 1957) stellten die gleichen 9 Länder 75% aller positiven Reaktionen.

3. Fünf Hauptpunkte verdienen besondere Aufmerksamkeit. Es sind dies:

1. Wir müssen uns unseres Vertrauen zur Tuberkulinprobe erneut versichern; ist sie doch bei sorgfältiger Ausführung und Ablegung anerkennt als eine der verlässlichsten Proben für die Entdeckung einer Erkrankung, die wir haben. Allzu häufig wird eine unangemessene Besorgnis zum Ausdruck gebracht, weil manche Tiere, die auf die Tuberkulinprobe reagieren, keine makroskopischen Befunde einer Tuberkulose bei der Autopsie aufwiesen. Das beste wissenschaftliche Beweismaterial und die Beobachtung an Millionen von Kadavern zeigen eindeutig, dass dem Tuberkulintext gegenüber den üblichen post mortem-Untersuchungen mehr Beweiskraft zukommt als einem Verfahren zur Feststellung der Tuberkulose.

2. Wir müssen der ordnungsgemässen Quarantäne von infizierten Herden sorgfältige Aufmerksamkeit zollen und uns die Gewissheit verschaffen, dass nicht ein als infiziert oder als exponiert bekanntes Tier die Ursache für die Vorbereitung der Krankheit wird.

3. Wir müssen uns darüber versichern, dass alle Baulichkeiten, in denen sich tuberkulöse Tiere aufgehalten haben, genügend gereinigt und desinfiziert werden unter peinlicher Überwachung.

4. Wir haben gewaltige Erfolge zu verzeichnen bei der Auffindung vieler Infektionszentren als Folge von epizoologischen Untersuchungen. Periodische Testung allein reicht nicht aus. Wir müssen alle Tiere, die bei regulärer Tötung tuberkulöse Befunde zeigen, zurückverfolgen bis zu ihren Heimatherden, und wir müssen die Herkunft von Tieren bestimmen, die auf Tuberkulinproben reagieren, und wir müssen die von infizierten Herden exponierten Tiere nachkontrollieren. Man muss mehr Anstrengung darauf verwenden, die Tuberkulose bei Geflügel und Schweinen auszumerzen.

5. Wir müssen wieder Geldmittel verteilen, um ein stärker konzentriertes Programm in denjenigen Staaten einzuschlagen, in denen jetzt die Mehrzahl der infizierten Tiere angetroffen wird.

Pulmonary Function Screening Tests In Bronchial Asthma

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Patients with bronchial asthma are frequently referred to pulmonary function laboratories for evaluation. It has been our custom to carry out routine screening tests. In addition a bronchial dilator drug has been administered to appraise the degree of bronchial spasm present. Review of our data reveals changes in the timed vital capacity, maximum breathing capacity and single breath nitrogen test which are the subject of this report.

Method

The routine screening tests comprise the following: (a) Spirographic record of vital capacity (VC) according to the method of Baldwin.[1] (b) Spirographic record of maximum breathing capacity (MBC) and determination of the presence or absence of elevation due to air trapping.[1] (c) Determination of timed vital capacity (TVC) by a method previously described.[2] The largest VC was used to calculate the timed vital capacity percentage. (d) Single breath O_2 test (SB), using a respirometer with attached potentiometer to record volume[3] and determination of terminal rise (SBTR) in the last 500 cc. of the alveolar nitrogen curve.[4] Isuprel 1:200 solution by nebulization was used to test for bronchial spasm.

Material

Twenty-eight patients with the diagnosis of bronchial asthma have been referred to the pulmonary function laboratory during the past 12 months. The majority were men ranging between 24 and 68 years of age with an average age of 50 years. The average duration of asthma was 11½ years with a range of four months to 38 years. These patients were divided into two groups: Group I, 19 who were receiving treatment for asthma in this hospital consisting of broad spectrum antibiotics, expectorants, bronchial dilator drugs and in some cases steroid therapy. Group II, nine with asthma who were tested prior to treatment immediately on admission to the medical wards.

Results

Table I gives the pulmonary function data on patients in the two groups. Table II gives data on the single breath oxygen test and the terminal rise on the alveolar nitrogen curve. Figure 1 shows a typical timed vital capacity test before and after the inhalation of Isuprel in a 57 year old man (J.J.S.) with history of asthma for 27 years. Figure 2 shows the single breath O_2 tests on the same patient.

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Discussion

Review of Table I shows that these asthmatic patients have an overall reduction in their vital capacities and maximum breathing capacities. These tests show improvement after Isuprel inhalation. Similar findings have been noted by others.[5-9] Elevation toward hyperinflation levels occurred in the maximum breathing capacity in 23 patients. In none of them was the elevated level significantly reduced after Isuprel. This would seem to indicate that a permanent type of air trapping has already developed. A similar finding suggested emphysema in non-asthmatic patients with pulmonary disease.[10]

Following the use of a bronchial dilator drug there is a striking apparent lack of improvement in the timed vital capacity when expressed as percentage. This finding is somewhat contrary to the expected result. The explanation is not far to seek: There is an actual increase in the rate of expiratory flow, but the increased volume in seconds is accompanied by an overall increase in vital capacity so that percentages frequently remain

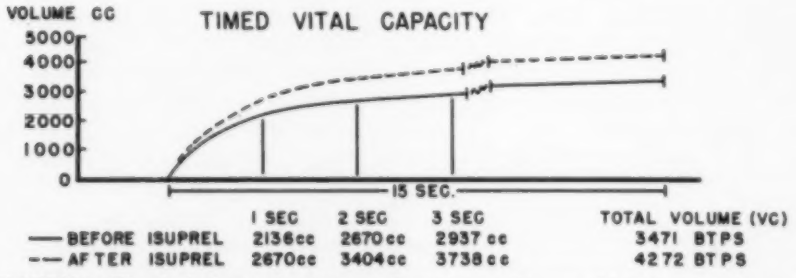

FIGURE 1: Timed vital capacity tests performed on a 57 year old male with diagnosis of bronchial asthma of 27 years duration.

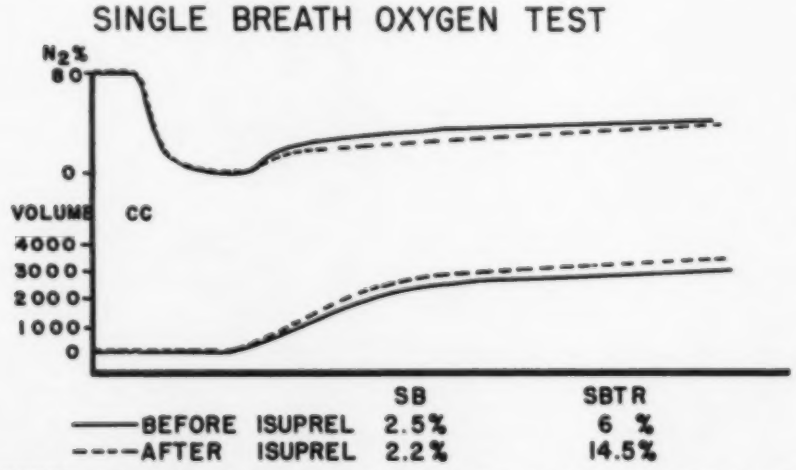

FIGURE 2: Single breath oxygen tests performed on a 57 year old male with diagnosis of bronchial asthma of 27 years duration.

unchanged and hence may be misleading. One may compare the curves (Fig. 1), record the volumes and compare the values directly, calculate percentage by dividing by the predicted vital capacity, or calculate the maximum expiratory flow rate. The mid expiratory flow rate described by Leuallen[11] is also unsatisfactory for demonstrating changes after Isuprel. The increase in volume tends to lower the midexpiratory portion of the curve into the slower range. The following example illustrates these points: A man 58 years of age gave a history of bronchial asthma for 37 years, VC before Isuprel 68 per cent, after Isuprel 82 per cent. TVC before Isuprel 1 second {29 per cent / 746 cc., 2 second {42 per cent / 949 cc., 3 second {50 per cent / 1356 cc.; after Isuprel 1 second {29 per cent / 1085 cc., 2 second {42 per cent / 1356 cc., 3 second {50 per cent / 1627 cc. Maximum midexpiratory flow before bronchial dilator 247 cc./second, after bronchial dilator 206 cc./second.

The nitrogen washout method was introduced in 1940 to test the uneven mixing of the respiratory gases within the lungs.[12] A number of investigations have shown that patients with asthma may demonstrate poor intrapulmonary mixing.[13-15] However, Comroe and Fowler[16] have shown that the single breath oxygen test is far superior in revealing poor distribution of gases in the asthmatic patient.[14, 16] They determined the index of intrapulmonary mixing on 19 asthmatic patients and found five to be

TABLE I
VENTILATORY TESTS ON 28 PATIENTS WITH BRONCHIAL ASTHMA

		GROUP I — 19 Hospitalized Patients Receiving Treatment				GROUP II — 9 Patients Tested On Day of Admission			
		Average vol. (cc.)	Per cent vol. change	Range per cent	TVC Percentage average	Average vol. (cc.)	Per cent vol. change	Range per cent	TVC Percentage average
VC	*AI	2878				3565			
	**PI	3126	+14	—15 to 58		4106	+18	—3 to +51	
TVC 1 sec.	AI	1311			43	2092			53
	PI	1498	+16	—13 to +45	44	2441	+22	0 to +47	54
2 sec.	AI	1731			58	2575			68
	PI	1994	+17	0 to +43	59	3013	+22	0 to +47	69
3 sec.	AI	1991			68	2834			75
	PI	2302	+18	0 to +37	68	3309	+22	+5 to +42	78
		L				L			
MBC	AI	47				72			
	PI	56	+17	+11 to +50		95	+42	+1 to +100	

*AI = Before Isuprel
**PI = After Isuprel

abnormal. On retesting 18 with the single breath test, 14 had poor intrapulmonary mixing. Our study shows that 26 in our group of 28 asthmatic patients had abnormal single breath oxygen tests values greater than 2 per cent. Twenty-seven were retested after Isuprel; three showed no change, 15 improved and 9 worsened. Table II gives the average changes in the single breath test and terminal rise before and after Isuprel administration. Review of our 27 patients on whom terminal rises were reported showed that 25 had abnormal values (greater than 4.5 per cent). Following bronchodilator 16 had terminal rises of greater magnitude. These changes in the terminal rise were more marked than seen in the first portion of the alveolar plateau (fig. 2). When worsening occurs in the single breath test or terminal rise it is probably due to opening poorly ventilated spaces into bronchi not communicating well until after dilation with Isuprel.

A comparison of Group I and Group II shows that asthmatic patients who have not been under recent treatment have the most striking changes after receiving a bronchial dilator drug. It would therefore appear that the appraisal of the effectiveness of bronchial dilator medication should be carried out on the day of admission rather than after the patient has received the usual therapeutic measures to alleviate the asthma.

The complication of emphysema is well recognized in patients with bronchial asthma. It is important to detect the development of this complication as early as possible. Attention is called to a recent study of the detection of early emphysema.[10] The same screening tests were used in the study of these asthmatic patients. The criteria for emphysema are a combination of prolonged timed vital capacity (two second less than 90 per cent), elevation of the maximum breathing spirogram toward hyperinflation level and abnormal single breath O_2 test (greater than 2 per cent). Applying these criteria to this series of patients with bronchial asthma it was found that 84 per cent had evidence of emphysema. Of this group only 70 per cent had x-ray interpretation of emphysema although PA and lateral films of the chest were available. The poor correlation of x-ray interpretation of emphysema with clinical findings has been commented upon by Knott.[17]

TABLE II
SINGLE BREATH OXYGEN TEST IN 28 PATIENTS WITH ASTHMA

		Average per cent	Average change per cent	Range of change per cent
SB (750-1,250 cc.)	*AI	5.7		
	**PI	5.4	+0.7 —0.3	—4.5 to +6
SB Terminal Rise (last 500 cc.)	AI	8.2		
	PI	9.5	+1.9 — +1.3	—3 to +9

*AI = Before Isuprel
**PI = After Isuprel

SUMMARY

(1) It is recommended that asthmatic patients be given the following routine pulmonary function screening tests: Vital capacity, timed vital capacity, maximum breathing capacity by the spirographic method and the single breath O_2 test with determination also of the terminal rise.

(2) If possible it is urged that asthmatic patients be tested with a bronchial dilator drug on admission to the hospital in order to demonstrate the degree of bronchial spasm. After the patients have been under treatment for several days the changes become less marked.

(3) Bronchial dilator drugs usually increase the vital capacity but the improvement may be greater in the maximum breathing capacity. Bronchial dilator drugs given to asthmatic patients do not have a significant effect on air trapping as demonstrated in the maximum breathing capacity spirogram.

(4) Bronchial dilator drugs appear to have little effect on the timed vital capacity if recorded in percentage values. Nevertheless, examination of the expiratory curve will show an increase in the volume per second and improvement in maximum expiratory flow.

(5) Bronchial dilator drugs may cause the single breath O_2 test to show worsening of the intrapulmonary mixing of gases. It is suggested that this change may be due to the opening of more poorly ventilated portions of the lung following bronchial dilitation.

(6) It is recommended that these screening tests be utilized to recognize the early development of emphysema so that preventive measures may be taken as soon as possible.

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RESUMEN

1. Se recomienda que los asmáticos se sometan a las siguientes pruebas de rutina: capacidad vital, capacidad vital en curva de tiempo, capacidad respiratoria máxima por método espirográfico, y la prueba simple de O_2 con determinación del alza terminal.

2. Si es posible se considera preciso que se pongan a prueba los asmáticos con un droga broncodilatadora, a su ingreso al hospital a fin de demostrar el grado de broncoespasmo. Después de que los enfermos han estado bajo tratamiento por varios días los cambios son menos marcados.

3. Las drogas broncodilatadoras generalmente aumentan la capacidad vital pero la mejoría puede ser mayor en la capacidad respiratoria máxima. Las drogas broncodilatadoras dadas a un asmático no tiene efecto de significación sobre el aire atrapado como lo demuestra el espirograma de la capacidad máxima respiratoria.

4. Las drogas dilatadoras parece que tienen poco efecto sobre la curva de tiempo de la capacidad vital si el registro se hace en valores porcentuales. Sin embargo el examen de la curva expiratoria mostrará un aumento del volumen por segundo y mejoría del flujo máximo expiratorio.

5. Las drogas broncodilatadoras pueden causar que la prueba simple

de O_2 respirado muestre empeoramiento de la mezola intrapulmonar de gases. Se sugiere que tal cambio se deba a la abertura de partes del pulmón más deficientemente ventiladas después de la dilatación bronquial.

6. Se recomienda que estas pruebas de rutina se empleen para reconocer tempranamente el desarrollo del enfisema de manera que las medidas preventivas se puedan poner en juego.

RESUME

1. L'auteur recommande de soumettre les asthmatiques aux tests habituels de la fonction pulmonaire tels que: capacité vitale, volume expiratoire maximal-seconde, ventilation maximale, par la méthode spirographique et le simple test de la respiration en oxygène, avec détermination également de l'élévation terminale.

2. Il est nécessaire si possible de soumettre les asthmatiques à un bronchodilatateur lors de l'admission à l'Hôpital, pour démontrer le degré du bronchospasme. Quand les malades ont été traités pendant plusieurs jours, les altérations sont moins apparentes.

3. Les médications broncho-dilatatrices augmentent généralement la capacité vitale, mais l'amélioration peut être plus grande en ce qui concerne la ventilation maximale. Les bronchodilatateurs donnés aux asthmatiques n'ont pas d'effet marqué sur la ventilation pulmonaire comme on peut le voir dans les tracés spirographiques de la ventilation maximale.

4. Les bronchodilatateurs semblent avoir un faible effet sur le volume expiratoire maximal seconde, si les résultats en sont rapportés en pourcentages. Néanmoins, l'examen de la courbe expiratoire montre une augmentation du volume par seconde et une amélioration du volume expiratoire maximum.

5. Les bronchodilatateurs peuvent, dans le simple test de la respiration en oxygène, être à l'origine d'une altération du mélange intrapulmonaire des gaz. L'auteur suggère que cette altération peut être due à l'apparition de parties pulmonaires plus pauvrement ventilées, par suite de la bronchodilatation.

6. Il recommande d'utiliser les tests qui permettent de reconnaitre le développement précoce de l'emphysème, afin de prendre les mesures préventives aussi rapidement que possible.

ZUSAMMENFASSUNG

1. Es wird empfohlen, dass bei asthmatischen Patienten die folgenden Lungenfunktionsproben angewandt werden: Vitalkapazität, begrenzte Vitalkapazität, Atemgrenzwert mittels der spirographischen Methode und der Sauerstoff-Atemtest mit Bestimmung auch des terminalen Anstiegs.

2. Es wird empfohlen, asthmatische Patienten, wenn möglich, zu testen mit einem bronchuserweiternden Medikament bei Krankenhausaufnahme, um das Ausmass des Bronchialspasmus zu bestimmen. Nachdem die Patienten mehrere Tage unter Behandlung gestanden haben, werden die Veränderungen weniger ausgeprägt.

3. Medikamente, die die Bronchien erweitern, vermehren gewöhnlich

die Vitalkapazität; doch kann die Besserung beträchtlicher sein für den Atemgrenzwert. Die Bronchien erweiternde Medikamente haben, wenn sie Asthmatikern gegeben werden, keine erhebliche Wirkung auf die Retension der Luft, wie sich anhand des Spirogrammes für den Atemgrenzwert nachweisen lässt.

4. Medikamente, die die Bronchien erweitern, scheinen wenig Wirkung zu haben auf die zeitbegrenzte Vitalkapazität, wenn sie in Prozentwerten aufgezeichnet wird, trotzdem wird die Auswertung des exspirotorischen Schenkels der Atemkurve eine Zunahme in Volumen pro Sekunde ergeben und eine Verbesserung im maximalen exspiratorischen Ausstoss.

5. Medikamente, die die Bronchien erweitern, können die Ursache dafür sein, dass der Sauerstoff-Atemtest eine Verschlechterung des intrapulmonalen Gasaustausches zeigt. Es wird angenommen, dass diese Veränderung die Folge eines Zugänglich-werdens schlechter ventilierter Lungenabschnitte sein kann in Folge Bronchuserweiterung.

6. Es wird empfolhen, dass diese Untersuchungsmethoden dazu benutzt werden, um die Entwicklung eines Emphysems frühzeitig zu ermitteln, so dass Präventiv-Massnahmen so zeitig wie möglich ergriffen werden können.

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Pleuropneumonectomy in Tuberculosis

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Introduction

Although the importance of completely removing an empyema wall was recognized many years ago by Fowler,[4] the recent increase in the direct surgical attack on tuberculosis has again drawn attention to this principle. Sarot's report[6] of two planned pleuropneumonectomies in 1947 in patients with tuberculosis and bronchopleural fistula gave great impetus to the recent development of this technique. Tchertkoff and Selikoff[7] confirmed the improved clinical results when the extrapleural approach was used in a review of 77 pneumonectomies at the Sea View Hospital. They found no death and no empyema in 25 extrapleural pneumonectomies while there were nine deaths and 11 bronchopleural fistulae in 52 patients operated by the intrapleural technique. Although this method has gained wide use recently, we have found only a few articles[2,3] dealing with the subject.

Since March of 1954, 38 pneumonectomies have been performed for tuberculosis at the Chicago State Tuberculosis Sanitarium. Thirteen were on the right and 24 were on the left. We have divided these patients into three groups according to the degree of pleural disease. In the first group, numbering nine cases, pleural adhesions were thin and band like, or there was a small area of fixation of the lung, usually in the apical posterior region to the pleural gutter. In the second group, in which there were 21 cases, the entire upper lobe or upper half of the lung was firmly fixed to the parietal pleura, requiring extra pleural separation. In the third group, in which there were eight cases, the pleural space was completely obliterated or replaced by empyema, necessitating total extrapleural resection. In this group were also included four patients with pre-operatively diagnosed bronchopleural fistulae.

Technique

The operative technique used in this series of cases is in principle similar to that which was previously described by Sarot.[5] The lateral, or occasionally, the face down position is used and the sixth rib usually excised subperiosteally. In the first group (those with the least amount of pleural fixation), the free pleural space is entered and adhesions separated bluntly, or, if necessary, divided with the scissors. If an area of firm fixation of lung to chest wall exists it usually lies in the posterior gutter. The extrapleural plane is entered at the edge of such an area by cutting directly into pleura against the rib cage. A finger is introduced and the pleura separated from chest wall by blunt or occasionally sharp dissection. The dissection is again carried intrapleurally by dividing the thin pleura around the circumference of the adherent portion. The parietal pleura thus freed is removed attached to the lung.

In the second group (those in which the upper half of the lung is largely involved in firm pleural adhesions), it is frequently possible to find a free or but lightly obliterated pleural space at the anterior end of the thoracotomy incision. Thin adhesions are separated by sharp or blunt dissection as indicated. Where the lung is firmly adherent, the extrapleural plane is entered as before. The extrapleural dissection is first carried anteriorly, by blunt finger dissection if possible, toward the sternal end of the superior mediastinum. When the mediastinum is reached the pleural space can usually be re-entered and the dissection carried in a posterior direction down the pleural surface of the mediastinum. Although these mediastinal adhesions tend to be less severe than chest wall adhesions,[5] this is not always true and it is sometimes necessary to carry the extrapleural plane all the way around to the posterior mediastinum. When the mediastinal adhesions are dense, one of the most difficult technical points is determining when to change the direction of dissection from the plane running medially along the chest wall to the plane running posteriorly along the mediastinal parietal pleura. If this is not done soon enough it is possibe to damage mediastinal structures or to enter the opposite pleural cavity.

During the mediastinal dissection on the left side, the aortic arch lies behind the dissecting finger while on the right side the area of dissection is along the azygos vein. On the left the phrenic and vagus nerves are palpable behind the dissecting finger as it progresses posteriorward, thus assuring that the plane of dissection is lateral to these structures. When the separation of lung and mediastinum is as complete as practicable from

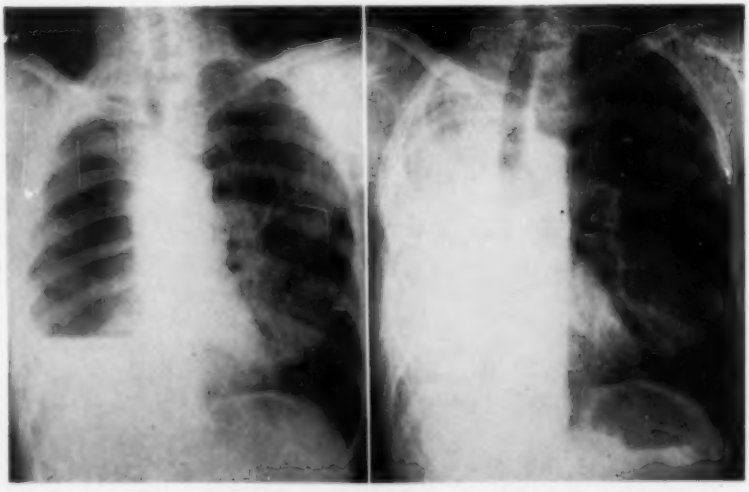

FIGURE 1A FIGURE 1B

Figure 1A: Preoperative chest x-ray of the first case of a patient with bronchopleural fistula and mixed empyema. The totally collapsed lung and large empyema space, occupying almost the entire pleural cavity are apparent.—*Figure 1B:* Chest x-ray following pleuropneumonectomy of patient shown in Figure 1A.

this direction, attention is directed to the posterior end of the thoracotomy incision. The extrapleural plane is then entered here, usually at or near the edge of the incision. Blunt dissection is carried medially behind the gutter, then anteriorly over the vertebral bodies until one breaks through into the field of dissection exposed from the front, or this dissection is carried as far as possible if this approach is elected as the starting point for the mediastinal dissection. At this point the operator has surrounded the lung just above its hilum, leaving the apex adherent to the dome of the pleura. These apical adhesions are then attacked in their full circumference working toward the cupola, by blunt and sharp dissection, in the extrapleural plane as required. The great vessels and other mediastinal structures are to be sought and avoided as the cupola is approached.

In the third group (those patients with a residual empyema or a densely obliterated pleural space), the dissection is completely extrapleural. The pleural space is never entered and the dissection is begun in the extrapleural plane at the wound edge. This plane is encountered after the periosteal rib bed has been divided. Posteriorly, the longitudinal fibers of the frequently present subcostal muscle lie between pleura and rib. The upper portion of the lung is separated in the extrapleural plane by the technique previously described. The extrapleural plane is then entered along the inferior edge of the thoracotomy incision and the dissection carried downward to the costophrenic angle where it is frequently very difficult to separate this greatly thickened peel. The diaphragmatic and mediastinal surfaces are separated likewise in the extrapleural plane, and the hilum finally approached. Although these empyema spaces can be removed intact using this technique, they will frequently be entered for convenience of

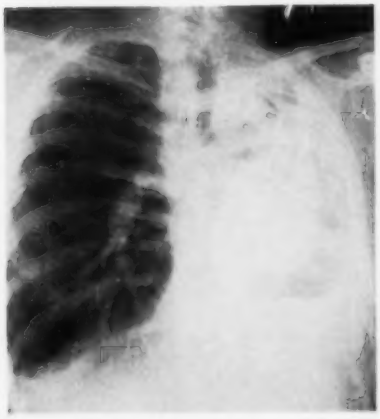

FIGURE 2A

FIGURE 2B

Figure 2A: The preoperative chest x-ray of the second patient with bronchopleural fistula. A thin line of air can be seen between the lateral border of the almost completely collapsed lung and the greatly thickened empyema wall. This represented the visualized portion of the empyema space.—*Figure 2B:* An illustration to show the empyema cavity in Figure 2B.

dissection or by accident. In the latter circumstance the remnants of the empyema wall are ultimately excised so that healthy raw muscle, fascia and periosteum only line the pleural cavity. By this method, even though the empyema content has been spilled and the surface of the healthy tissue contaminated, all of the empyema wall in which the tuberculous infection is actively established is removed.

Clinical Reports

The eight patients in whom total extrapleural pneumonectomy was performed will be considered in more detail. Five were on the right and three were on the left. Six of these patients had good, one fair and one had poor antimicrobial drug coverage. Preoperatively, four of the eight had bronchopleural fistulae, and these patients were particularly difficult problems.

The first was a 57 year old white woman with an 18 year history of tuberculosis. She had had therapeutic pneumothorax at the onset which had been followed by chronic empyema of 16 years duration. Her right lung was completely collapsed (Figure 1 A). Her vital capacity was 840 cc. (32 per cent of normal) and her maximum breathing capacity was 25 liters per minute (40 per cent of normal). Her post pneumonectomy course was uncomplicated and she filled her hemithorax satisfactorily (Figure 1 B).

The second case was that of a 37 year old white man with tuberculosis of 19 years duration, for whom pneumothorax had been performed at the onset. His bronchopleura fistula was diagnosed by the presence of a thin rim of air between the lateral surface of his greatly collapsed lung and the

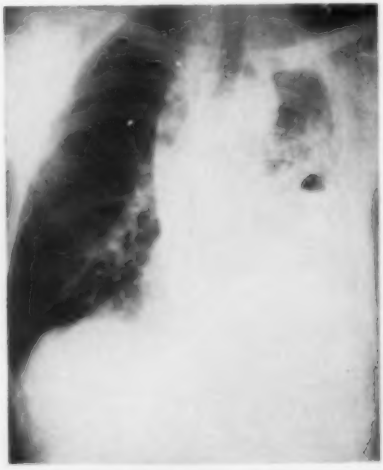

FIGURE 3A

FIGURE 3B

Figure 3A: The preoperative chest x-ray of the third patient with bronchopleural fistula. This case was remarkable because of the degree of calcification of the empyema wall which had taken place.—*Figure 3B:* An illustration of the x-ray in Figure 3A. The shaded area indicates the location of the calcium.

greatly thickened pleural peel (Figure 2 A). His vital capacity was 1,340 cc. (31 per cent of normal) and his maximum breathing capacity was 38 liters per minute (21 per cent of normal). He had persistently positive sputum on concentrated smear and the organism was highly resistant to streptomycin, para-aminosalicylic acid and isoniazid. He was placed on viomycin and P. Z. A. ten days before operation. Postoperatively he required oxygen for several weeks; however, he gradually regained sufficient respiratory reserve to obviate this and became freely ambulatory.

The third case was that of a 64 year old white man whose disease was of unknown duration, and in whom a marked amount of calcium lined the empyema cavity at surgery (Figure 3). The procedure was difficult because of the extension of this calcified peel into the chest wall.

The fourth case was that of a 55 year old Negro with a 10 year history of tuberculosis which had been treated by pneumothorax at the onset. He had a vital capacity of 2,430 cc. (67.5 per cent of normal) and a maximum breathing capacity of 42 liters per minute (46.5 per cent of normal). His empyema cavity was outlined preoperatively by a bronchogram (Figures 4 A and B). He developed an empyema which required open drainage six months postoperatively.

Of the four patients requiring pleuropneumonectomy who did not have bronchopleural fistulae, one had had a previous wax plombage thoracoplasty. The wax was removed at the time of pleuropneumonectomy. The chest wall between thoracoplasty space and pleural space was left intact although the lung was removed extrapleurally. He developed postoperative empyema requiring open drainage, as did one other patient. Thus, of the

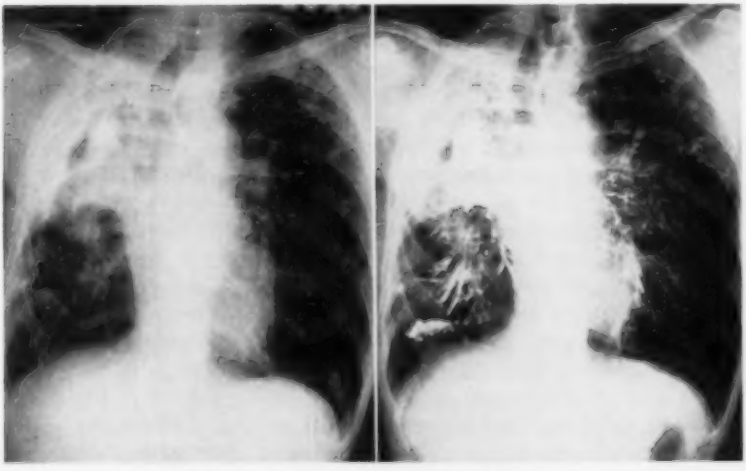

FIGURE 4A FIGURE 4B

Figure 4A: The preoperative chest film of the fourth patient with bronchopleural fistula. A fluid level may be seen low in the right lung field. No cavity was demonstrable in the left lung.—*Figure 4B:* A bronchogram of the fourth patient showing contrast medium "layering" on the surface of fluid levels within the empyema loculations. This demonstrates the presence of a bronchopleural fistula.

eight in whom extrapleural pneumonectomy was performed, three developed postoperative empyema (37 per cent). Two of these empyemata occurred in the group without and one in the group with preoperative bronchopleural fistula. None in this series developed postoperative bronchopleural fistula.

In general these procedures are tedious and difficult because of the tough adhesions and distorted anatomy. Occasionally it even seems questionable whether or not they can be carried on to completion. As has been noted frequently in these cases of long standing pleural disease the ribs are altered from their usual flattened to a triangular contour due to the addition of a wedge like extension of bone from along their inner surfaces. This is presumably a periosteal response to inflammation during the early stage of the pleural disease and a mechanical counter reaction to the shrinking tendency of the scarred pleura in the late stages. The latter is a manifestation of Wolff's law, according to Bisgard[1] who has investigated this interesting phenomenon. "Shelving" or "shingling" of the ribs likewise may be noted as a result of shrinkage of the chest wall over the diseased pleura. In some cases there is partial calcification of the peel and it may extend into the intercostal space so that it is necessary to use a periosteal elevator to separate the tuberculous peel from the underlying muscle and ribs to which it is so firmly fixed. These procedures usually require blood replacement.

The immediate postoperative condition of all the patients in this series was good considering the magnitude of the procedure. One noticeable finding was an increase of neck and chest wall emphysema, often with associated nasal speech (Rhinolalia). This was obviously due to the fact that when the parietal pleura is stripped, muscle and fascial planes are opened toward the mediastinum and neck, into which air from the empty hemithorax is more easily forced.

SUMMARY AND CONCLUSIONS

In 38 pneumonectomies performed at the Chicago State Tuberculosis Sanitarium during the past three years, the extrapleural plane of dissection was utilized over whatever areas of lung were firmly fixed to chest wall. Of these 38 patients, eight were done by the total extrapleural method. Four of these eight had preoperative bronchopleural fistulae with mixed empyemata. Antituberculosis drugs have made more patients amenable to surgery and have stimulated interest in this technique.

Empyema following pneumonectomy is a cause of greater morbidity than that following lobectomy, where lung tissue remains to eventually fill the pleural space. Among patients requiring pneumonectomy for tuberculosis, who have also had tuberculous pleuritis and especially empyema, it is of the greatest importance to remove all the pleural peel. This is infected tissue, in which the tubercle bacillus is firmly established. If left behind, it represents a continuous source of contamination for the dead space within the hemithorax. The removal of this peel is most easily accomplished by developing the extrapleural plane as completely as possible early in the procedure and if feasible, removing the empyema completely

without entering its cavity. Preserving the integrity of the empyema, however, is not as important as removing the entire empyema wall, since the surface of relatively normal tissue remaining after extrapleural dissection can withstand a single contamination by empyema fluid, whereas it may not withstand the continuous contamination by remaining infected tissue.

From the technical standpoint, the extrapleural plane, if properly developed, is safe, and in patients with marked pleural disease, often the easier plane of dissection. In addition, one is less apt to break into thin-walled apical cavities with the added thickness of the pleura between the lung and dissecting finger. With this technic, bleeding may actually be less than in procedures where cut surface of peel is left *in situ*, for vessels in normal tissue retract more certainly than those developed as a result of inflammation.

RESUMEN Y CONCLUSIONES

En 38 neumonectomías realizadas en Sanatorio Estatal de Chicago durante los tres años pasados, se usó el plano extrapleural para la disección ante cualquiera área del pulmón muy adherida a la pared.

De estos 38 enfermos, en ocho se hizo todo el procedimiento por vía extrapleural. Cuatro de estos ocho tenían fístula broncopleural con empiema mixto preoperatoriamente. Las drogas antituberculosas han hecho más enfermos adecuados para la cirugía y han estimulado el interés en esta técnica.

El empiema después de la neumonectomía es una causa de mayor morbilidad que en el caso de la lobectomía en la que el tejido pulmonar restante eventualmente llena el espacio pleural. En los enfermos qu necesitan neumonectomía por tuberculosis, que tienen también pleuritis tuberculosa y especialmente empiema es de la mayor importancia quitar toda la pleura. Esta constituye tejido infectado en el bacilo de la tuberculosis que está firmemente establecido.

Si se deja la pleura esto representa un peligro constante de contaminación del espacio muerto. La extirpación de la pleura es más fácilmente lograda desde el principio del procedimiento buscando desde luego el plano extrapleural y si es posible quitando el empiema completamente sin entrar en su cavidad. El conservar la integridad del empiema sin embargo, no es tan importante como quitar toda la pared de él puesto que la superficie de tejido relativamente sano quo se deje después de la disección extrapleural, puede soportar una simple contaminación por el líquido empiemático en tanto que no resiste a la continua contaminación de tejido infectado que se deje.

Desde el punto de vista técnico el plano extrapleural si se aborda bien, es seguro y en enfermos con seria enfermedad pleural a menudo el plano más fácil de disecar.

Además uno puede menos fácilmente romper la pared pleural y caer sobre cavernas de pared muy delgada cuando se agrega la pleura que se diseca por afuera con el dedo. Con esta técnica el sangrado de hecho puede ser menor que en los procedimientos en que una parte cortada de la pleura

se deja porque los vasos del tejido normal se retraen más que los desarrollados como consecuencia de la inflamación.

RESUME

Au cours de 38 pneumonectomies pratiquées au Sanatorium de l'Etat de Chicago pendant les trois dernières années, on utilisa le plan extrapleural de dissection, même si des zones de poumon étaient fermement fixées à la paroi thoracique. Sur ces 38 malades, 8 furent opérés par la méthode extrapleurale totale. 4 de ces huit malades étaient porteurs avant l'opération de fistules bronchopleurales associées à des épanchements Les médications antituberculeuses ont permis d'opérer un plus grand nombre de malades et ont augmenté l'intérêt de cette technique.

L'épanchement qui fait suite à une pneumonectomie est une complication plus sévère que celui qui survient après une lobectomie, car il reste alors du tissu pulmonaire qui peut éventuellement combler l'espace pleural. Parmi les malades justiciables d'une pneumonectomie pour tuberculose, qui ont eu également une atteinte pleurale et particulièrement un épanchement, il est de la plus grande importance d'enlever tous les feuillets pleuraux. Il s'agit de tissu infecté, dans lequel le bacille tuberculeux est fermement établi. Si on le laisse en place, il représente une source permanente de contamination pour l'espace mort de l'hémithorax. L'xérèse de cette plèvre est plus facilement accomplie en développant le plan extrapleural aussi complètement que possible, dès le début de l'opération, et si c'est réalisable, en asséchant complètement l'épanchement sans entrer dans sa cavité. Préserver l'intégrité de l'empyème cependant, n'est pas aussi important que d'enlever la paroi complète de l'empyème, puisque la surface de tissu relativement normal restant après dissection extrapleurale peut s'opposer à une simple contamination par épanchement liquide, alors qu'elle ne peut pas s'opposer à la contamination permanente par le tissu infecté restant.

Du point de vue technique, l'intervention dans le plan extrapleural, si elle est convenablement exécutée, est sans danger, et chez les malades atteints d'affection pleurale grave, c'est souvent le plan de dissection qu'on peut suivre le plus facilement. En outre, le chirurgien risque moins d'entrer dans les cavernes apicales à parois fines s'il s'ajoute l'épaisseur de la plèvre entre le poumon et le doigt qui dissèque. Avec ce procédé, le saignement peut être moindre que dans les procédés où on laisse en place une portion de la zone de section, car les vaisseaux du tissu normal se rétractent plus surement que ceux qui si sont développés par suite de l'inflammation.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNGEN

Bei 38 im Staatlichen Tuberkulose-Sanitarium von Chicago während der verflossenen 3 Jahre durchgeführten Pneumonektomien wurde die extrapleurale Lösungsschicht ausgenutzt ohne Rücksicht darauf, welche Lungenabschnitte fest mit der Brustwand verwachsen waren. Unter diesen 38

Kranken wurde bei 8 die totale extrapleurale Methode angewandt. 4 von diesen 8 hatten vor der Operation eine innere Fistel mit Mischinfiziertem Empyem. Die antituberkulösen Heilmittel ermöglichten es, dass mehr Patienten auf chirurgischem Wege angegangen werden konnten und haben das Interesse an dieser Operationstechnik angespornt.

Empyeme in Anschluss an Pneumonektomien sind häufiger die Ursache von Morbidität als nach Lobektomien, wobei Lungengewebe zurückbleibt, um gegebenenfalls die Pleurahöhle auszufüllen. Bei Kranken, die einer Pneumonektomie wegen Tuberkulose bedürfen und die auch eine tuberkulöse Pleuritis und vor allem ein Emypem gehabt haben, ist es von grösster Wichtigkeit, den gesamten pleuralen Sack zu entfernen. Hier handelt es sich um infiziertes Gewebe, in dem der Tuberkelbazillus fest eingenistet ist. Wird er zurück gelassen, stellt er eine ständige Quelle der Verunreinigung dar für den toten Raum innerhalb der Thoraxhälfte. Die Entfernung dieses Sackes geschieht am leichtesten durch Darstellung der extrapleuralen Schicht und zwar so vollständig wie möglich schon im Anfang des Eingriffes; und wenn möglich, entfernt man das Empyem vollständig, ohne in seine Höhle einzudringen. Das Empyem unversehrt zu erhalten, ist jedoch nicht von derselben Wichtigkeit wie die Entfernung der gesamten Eympemwand, denn die Oberfläche des relativ normalen Gewebes, das nach der extrapleuralen Auschälung zurückbleibt, kann wohl einer einmaligen Verunreinigung durch die Empyemflüssigkeit Widerstand leisten, nicht aber der fortgesezten Verunreinigung durch das zurückgelassene infizierte Gewebe.

Vom technischen Standpunkt ist die extrapleurale Schicht, wenn sie ordnungsgemäss dargestellt wird, sicher und häufig bei Kranken mit ausgeprägten pleuralen Erkrankungen die frühzeitigere Durchtrennungsschicht. Darüber hinaus widerfährt es einem weniger leicht, in dünnwandige Spitzencavernen zu perforieren dank hinzugekommener Verdickung der Pleura zwischen der Lunge und dem lösendem Finger. Mit dieser Technik kann die Blutung tatsächlich geringer sein, als bei Metoden, bei denen die druchtrennte Oberfläche der Schwarte *in situ* belassen wird denn Gefässe in normalen Gewebe ziehen sich mit mehr Gewissheit zurück als solche, die entstanden sind im Gefolge einer Entzündung!

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Segmental Anatomy as Applied to Segmental Resection

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The concept of the lung as being composed of 10 bronchopulmonary segments on each side rather than two or three lobes has extended the scope of thoracic surgery.[1-3] The chest surgeon now carries out planned segmental resections to eradicate pulmonary pathology with the conservation of a maximum of healthy parenchyma. By definition, segmental resection is the removal of an intact solitary segment by anatomically isolating its blood supply and bronchus and after severing these structures stripping out the segment in toto. This is in contradistinction to wedge excision which has no respect for anatomical nicety, although indicated at times, particularly for peripheral lesions. To devitalize and defunctionalize parenchyma by lack of knowledge of its segmental blood supply relegates the involved area to category of mere "stuffing." To remove uninvolved segments that can be saved is sheer waste of needed alveoli. At the same time the surgical limitations of segmental resection should be recognized and are not as widely appreciated as they should be.

Several diseases have definite segmental importance. One of these is bronchiectasis which starts in one or more segments and spreads to another segment only with the advent of another pneumonic episode, so frequent in this disease. The extent of the disease can be readily determined by bronchography and rather neatly removed by segmental resection. Many benign neoplasms and granulomas can be so removed. When a bronchial adenoma occurs, in one out of 10 instances it is located in a segmental bronchus rather than a main bronchus. The relatively recent understanding of the nature of obstructive pneumonitis has given us this all important, often initial, diagnostic indication of bronchial neoplasm, often in a segmental bronchus.

The bronchopulmonary segments can be classified as resectable individually, resectable with difficulty and inadvisable to resect.

Considering the crossing of intersegmental lines by arteries, veins and subsegmental bronchi, there are three segments on each side resectable individually in most instances (Fig. 1): the anterior segment (B^2) of the upper lobe and the superior (B^6) and medial (B^7) of the lower lobes. Resectable with difficulty individually are three segments in each lung (Fig. 2): the apical (B^1) and posterior (B^3) of the upper lobe and the anterior basal (B^8) of the lower lobe. Inadvisable to resect individually are (Fig. 3): the segments of the lingula and the middle lobe (B^4 and B^5), the subsuperior (B^*), the lateral (B^9) and the posterior basal (B^{10}) of the lower lobes; the first (B^4 and B^5) because of the very common intersegmental

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crossing and the latter three (B^8, B^9 and B^{10}) because of inaccessibility of segmental vessels and bronchi.

Planned segmental resections vary in extent from resection of one segment, such as resection of the left medial basal segment (B^7) (Fig. 4) to resection of several segments (Figs. 5 and 6).

If one compares the relative ease of resection of the anterior segment (B^2) of the right with surgical hazards of resection of the apical (B^1) or

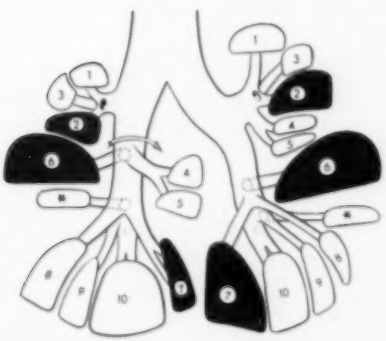

FIGURE 1

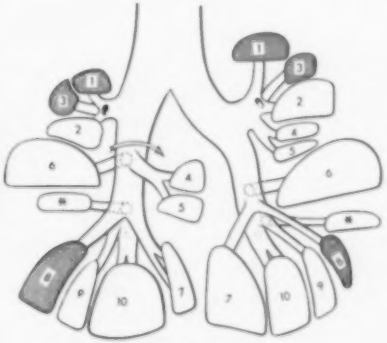

FIGURE 2

Figure 1: In each lung the anterior (B^2), superior (B^6) and the medial basal (B^7) segments can be resected individually with ease. Above is a sketch showing diagrammatically the segments of the lung. The middle lobe is swung medially out of position to show more closely all of the segments of the right lung.—*Figure 2:* In each lung the apical (B^1), posterior (B^3) and the anterior base (B^8) segments are resectable with difficulty.

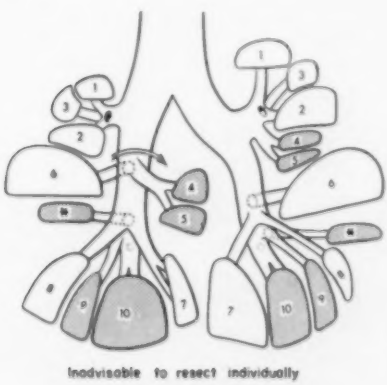

FIGURE 3

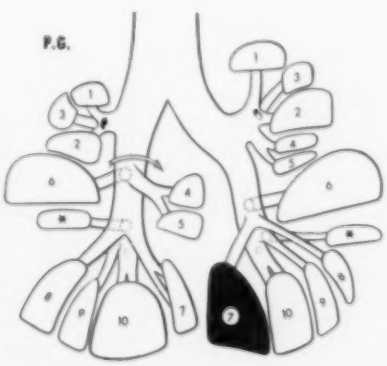

FIGURE 4

Figure 3: The lingula (B^4, B^5), the middle lobe (B^4, B^5), the subsuperior (B^8), the lateral basal (8^9) and the posterior basal segments are inadvisable to resect individually due to very common intersegmental crossing and inaccessibility of the segmental bronchi and vessels.—*Figure 4:* This sketch (P.G.) demonstrates resection of only the left medial basal segment for bronchiectasis.

posterior (B^3), one is impressed by the difference. Resection of the anterior segment (B^2) interrupts the blood supply of adjacent segments in only one out of three lungs. However, resection of either the apical (B^1) or the posterior (B^3) segment constitutes much more complicated problems. The apical segmental artery (A^1) or the posterior segmental artery (A^3) cross intersegmental lines in slightly less than one out of three lungs. Further, these two arteries arise from a common stem in three out of four lungs. In addition there is the hazard in one out of three cases of a split posterior segmental bronchus (B^3) with the posterior subsegmental bronchus (B^3b) arising from the anterior bronchus (B^2) and the apical subsegmental bronchus (B^3a) arising from the apical segmental bronchus (B^1).

The right medial basal segment (B^7) deserves particular attention as this is the only predictable segment as far as resectability is concerned (Figs. 4 and 5). If by bronchography one can see that the subsegmental bronchi are rather wide apart, one can presume that it is one of the 26 per cent of instances where the vein is straddled by the subsegmental bronchi and resection of this segment is not usually going to be possible.[3] This vital anatomical fact should affect very substantially decision concerning planned segmental resection.

Of particular interest is the case (Fig. 6) of a 34 year old Scandinavian woman with a 30 year history of bronchiectasis for whom I resected all of the segments of the right lung except the apical and posterior segment of the right upper lobe and the superior of the lower lobe, making a new lung out of the remainder with the reduction of sputum of 95 per cent. Bilateral resections of bronchiectatic segments have also been done with marked improvement.

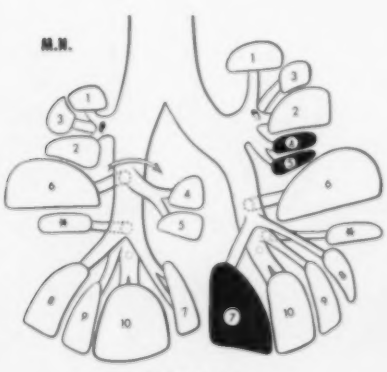

FIGURE 5

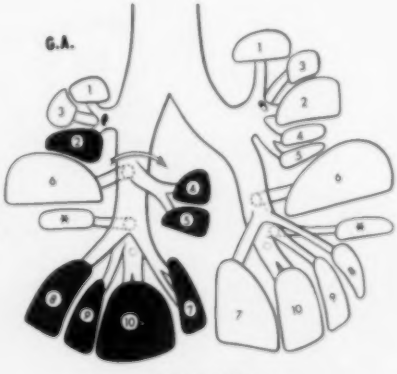

FIGURE 6

Figure 5: This sketch (M.N.) shows resection of the lingula and the left medial basal segment for bronchiectasis.—*Figure 6:* This sketch (G.A.) demonstrates multiple segmental resections for bronchiectasis. The anterior segment, middle lobe and the lower basal segments were removed. A new "lung" was constructed of the apical, posterior, superior and subsuperior segments with gross reduction of sputum. The other lung, if also diseased, can be similarly handled.

SUMMARY

True segmental resection is not a simple matter and there is need to redefine accurately the term. Knowledge and employment of anatomy are necessary to avoid devitalization of some segments and unnecessary removal of other segments. The segmental anatomical hazards must be understood to perform accurate segmental resections.

The limitations of segmental resection must be appreciated as well as the advantages.

There are three segments readily resectable individually on each side, the anterior, superior and medial basal; there are three segments resectable with difficulty individually, the apical, posterior and anterior basal; and the segments inadvisable to resect individually are the segments of the lingula and middle lobe, the subsuperior, the posterior basal and lateral basal.

RESUMEN

La verdadera resección segmentaria no es una cosa tan sencilla y es necesario revisar la definición del término.

Es necesario conocer y emplear conocimiento, de la anatomía, para evitar la desvitalización de ciertos segmentos así como la extirpación innecesaria de otros. Los azares anatómicos deben comprenderse para realizar las resecciones segmentarias. Deben tenerse en cuenta las limitaciones y las ventajas de la resección segmentaria.

Hay tres segmentos fácilmente resecables individualmente en cada lado: el anterior, el superior y el medial basal. Hay tres segmentos que son resecables difícilmente por separado: el apical, el posterior y el anterior basal; y los segmentos que no son aconsejables para resecar individualmente son: Los de la língula, y del lóbulo medio, el subsuperior, el posterior basal y el lateral basal.

RESUME

La résection segmentaire n'est pas une notion simple et il est nécessaire de définir de nouveau le terme avec précision. La connaissance et l'utilisation de l'anatomie sont nécessaires pour éviter la dévitalisation de certains segments et l'excision inutile d'autres segments. Les anomalies anatomiques segmentaires doivent être connues pour pratiquer des résections segmentaires précises.

Les limites de la résection segmentaire doivent être appréciées ainsi que ses avantages.

Il y a trois segments de chaque côté qui peuvent être réséqués sans difficulté à titre isolé: l'antérieur, le supérieur, et le moyen basal. Il y a trois segments qui peuvent être isolément l'objet d'une exérèse mais au prix de certaines difficultés; ce sont: l'apical, le postérieur et l'antérieur basal. Les segments dont la résection isolée est déconseillée sont les segments de la lingula et du lobe moyen, le sous-supérieur, le postérieur basal, et le latéral basal.

ZUSAMMENFASSUNG

Eine regelrechte Segmentresektion ist keine einfache Angelegenheit, und es besteht keine Notwendigkeit, den Ausdruck noch einmal genau zu definieren. Kenntnis und Gebrauch der Anatomie sind erforderlich, um zu vermeiden, einige Segmente ihrer Lebenskraft zu berauben und unnötiger Weise andere Segmente zu entfernen. Die dem Segmente inne wohnenden anatomischen Zufälligkeiten muss man verstehen, um sorgfältige segmentale Resektionen vorzunehmen. Die Abgrenzugen der segmentalen Resektionen müssen ebenso richtig eingeschätzt werden, wie ihre Vorzüge.

Es gibt auf jeder Seite 3 einzelne, bequem zu resezierende Segmente, nämlich das anteriore, das superiore und das medio-basale; und es gibt 3 Segmente, die einzeln mit Schwierigkeiten zu resezieren sind das apicale, das posteriore und das antero-Basale; und die als einzelne besser nicht zu resizierenden Segmente sind diejenigen der lingula und des Mittellappens, das subsuperiore, das posterior-basale und das latero-basale.

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Broncho-Pulmonary Amoebiasis*

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Introduction

Amoebiasis is mainly and primarily an infection of the colon by the pathogenic amoeba, Endamoeba histolytica, but it is also characterised by a variable clinical course of which the manifestations caused by the occurrence of complications may dominate the picture.

Invasion of the submucosa may be followed by entery of E. histolytica into the radicles of the portal vein and metastasis of the infection to the liver. This is followed by amoebic hepatitis or amoebic abscess of the liver which is by far the most frequent and important complication. However, it has always been known that the lung could be involved and that if the physician, particularly in an endemic area like ours, is amoebiasis conscious, he will be able to find out that amoebic infection is the real cause of so many cases that would have, otherwise, been diagnosed as pneumonitis or lung abscess of another undetermined or unknown etiology.

If we review the literature, we find that, as far back as 1873, cases started to be recorded, first by Colin and by Nexon, later, by Opie in 1901 and then Bunting in 1906 described a route by which endamoeba gain the pulmonary circulation and that was by direct embolism through the circulation from the colon and in this case firm consolidated nodules were found in the lung in which endamoeba were demonstrated in section. Since then, the literature is full of cases described clinically or in autopsies. In Egypt Girgis reported two autopsies in 1939 each of which showed multiple small abscesses in the right lung complicating active amoebic dysentery with multiple liver abscesses. Higazi (1945) reported one case of amoebic abscess of the right lower lobe of the lung complicating amoebic hepatitis and Gomaa (1946) reported three cases of amoebic lung infection as a cause of hoemoptysis. Abdel-Shafi (1952) published a synopsis of the morbid anatomy and histopathology of amoebiasis in which he recorded its manifestations in the chest in 25 autopsies.

Incidence

According to Roger, in a post mortem study of 81 fatal cases of amoebic abscess of the right lobe of the liver, no less than 26 had a secondary abscess in the lung, i.e., an incidence of about 32 per cent, but in the living, he reported that in a Calcutta series of liver abscesses in Europeans 20 per cent opened through the lungs, nearly invariably the right.

Abdel-Shafi, studied the post mortem records of 1000 cases autopsied in the pathology department, among which he met with 40 cases of

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amoebiasis. The incidence of amoebic chest manifestations, in all its forms, was 60 per cent but that of abscess formation in the lungs was 42.5 per cent. El-Mofti and Mousa (1951) analysed 900 cases of amoebic infection of which 50 manifested hepatic amoebiasis. Among these they reported that the lungs and the pleura were involved in 2 and 21 cases respectively.

In our experience, which is mainly based on clinical evidence it seems that broncho-pulmonary amoebic manifestations are not common and we think it is now less often seen than it was some years ago, possibly because of the more efficient treatment and better medical care offered the population and especially the peasants.

Route of Infection

Different modes of infection have been described in the literature, apart from the well known direct method of extension from the liver to the lung through the diaphragm.

There are cases which have been recorded, mainly by Harrington (1930) where lung abscesses of amoebic origin were detected in the upper lobe or presented as multiple small ones in both lungs and hence the possibility of blood borne infection from the liver into the inferior vena cava to the right side of the heart and through the pulmonary arteries to the lung. In fact in the two autopsies reported by Girgis, to which we have already referred, histological findings did reveal endamoeba in the emboli contained in the pulmonary arterial branches as well as in the abscess cavities.

The following are the possible routes of infection:

1. Primary pulmonary amboebiasis: where endamoeba reach the lung by direct embolism from the bowel through the middle and inferior hemorrhoidal veins. In these cases pulmonary amoebiasis may develop independently of hepatic affection and moreover the pulmonary condition does not necessarily manifest as suppuration but it may simulate bronchopneumonia or miliary tuberculosis. Autopsy of such cases revealed firm consolidated nodules in the lung in which endamoeba were demonstrated in the section.

2. Secondary pulmonary amoebiasis: This is the commonest variety. The condition is associated with or can be related to an amoebic liver disease. In such cases infection spreads from the intestines to the liver through the portal circulation and then reaches the lung:

    a. by direct extension through the diaphragm,

    or b. by embolisation from thrombosed hepatic veins to the inferior vena cava to the right side of the heart and through the pulmonary arteries to the lungs.

Recently cases of lung suppuration have been reported, caused by endamoeba gingivalis, a non-pathogenic saprophyte, which may be aspirated into the bronchial tree and produce disease.

Pathology

In primary cases, amoeba after reaching the lung, produce focal pneumonic consolidation soon followed by necrosis and softening as in the liver. With the discharge of the necrotic tissue into the pleural space or through the bronchi, a cavity is formed and the lobe undergoes, in part or all, a process of consolidation. Microscopically, there is an inner zone of necrotic pulmonary tissue and about this, the alveolar septa show marked broadening by interstitial fibrosis. The alveoli are small, lined with cuboidal epithelium and filled with vacuolated macrophages, lymphocytes and occasional plugs of fiibrin. The amoeba are found about the necrotic zone and may enter the bronchi and thus appear in the sputum.

Some have thought that suppuration may commence between the diaphragm and the liver or in the base of the lung but extensive Calcutta experience as reported by Roger shows that this is rarely, if ever, the case. What happens is that the capsule of the liver becomes fused with the diaphragm and the pus escapes into the surrounding tissues to such an extent that the primary liver abscess cavity may shrink to the size of the tip of a finger and escape observation while a small opening leads into a large secondary abscess in the base of the lung or between the liver and the diaphragm.

Clinical Manifestations

These depend on the type of pulmonary involvement whether it is primary and then, as mentioned above, simulating bronchopneumonia or tuberculosis, or secondary to rupture and then giving the characteristic suppurative syndrome. It may happen that the original hepatic abscess produced no symptoms whatever and therefore remained unrecognized till the rupture has taken place.

The diagnosis is more difficult in those cases belonging to the primary pulmonary group. It is based upon the previous evidence of amoebiasis, the leukocytosis and the remarkable and lasting response to emetine. As Manson Bahr mentions, it would have been more satisfactory to find E. histolytica in the sputum but from the nature of the lesions, which probably, as in Bunting's case, are composed of consolidated bronchopneumonic nodules, one would not believe that to be possible.

As with other parasitic infestations, their possible relation to any allergic manifestations needs consideration and in fact, the problem is discussed by Carri, in an interesting report from Argentina, concerning intestinal amoebiasis and respiratory allergy. The author notes that among 120 cases who were passing E. histolytica or its cysts, there were 12 who suffered from symptoms of respiratory allergy, three with spasmodic attacks of asthma and nine with other allergic symptoms. The cases were investigated and skin tests done with varying results. The usual remedies were used and anti-spasmodics tried with perhaps temporary but no permanent benefit. Emetine was prescribed followed by yatren and in each case improvement soon took place and was maintained.

To evaluate or criticise this report, more detailed information is re-

quired and a long period of observation is necessary. Anyhow, in our series we have not met with such cases.

Radiological Findings

The first changes are limited to an elevation of the right leaf of the diaphragm and restriction of its mobility. A localised bulging of the right diaphragm into the lower right lung field denotes a solitary amoebic liver abscess. Chest involvement comprises obliteration of the costophrenic sinuses, pleural effusion and pneumonic consolidation with or without abscess formation. In many cases, the roentgenograms were reported to show a string-like shadow proceeding perpendicularly from the localised bulge of the diaphragm to the pneumonic shadow in the lung. In view of the fact that these linear shadows were observed in cases of proved amoebiasis and disappeared after anti-amoebic treatment, it may be assumed that they are indicative of the spread of the inflammatory process. Such shadows when combined with other x-ray signs should induce the roentgenologist to consider the possibility of amoebiasis.

El-Deeb (1951) described among the radiological manifestations of amoebiasis of the lungs the presence of transverse bands of focal atelectasis in the lower lobe, a feature which we have also observed in two cases in our series.

Present Investigation

Our series includes 28 cases with amoebiasis manifested in the chest in the following forms:

Amoebic lung abscess	9 cases
Amoebic pneumonitis	9 cases
Amoebic bronchitis	2 cases
Pleurisy with serous effusion	5 cases
Empyema	3 cases
TOTAL	28 cases

Combined lesions in the chest were present in seven cases as follows:

Pleurisy with serous effusion, associated with:	
a—Amoebic pneumonitis	6 cases
b—Amoebic bronchitis	1 case

Sex incidence was highly prevalent among men, only two cases were women.

The youngest patient in our series was 21 and the eldest was 54 years old.

12 cases were in the third decade,

12 cases were in the fourth decade,

3 cases were in the fifth decade,

1 case was in the sixth decade.

So the commonest age incidence, was in the third and fourth decades (85.7 per cent of the cases).

These findings agree with the sex and age incidence of amoebic suppurative hepatitis reported by Roger as a result of the analysis of about 400 liver abscess cases in Calcutta which showed a marked preponderance in

men accounting for about 95 per cent of the cases and that the highest age incidence was between 21 and 40 years (70 per cent).

History

The duration of chest symptoms before coming for consultation varied between one and six months, the shortest period was 10 days and the longest was three years.

It was noticed that history of previous dysentery was present in 12 cases (43 per cent). The period that elapsed between the last complaint of dysenteric symptoms and the onset of chest manifestations varied between four months and one year. In most cases it was chronic amoebic dysentery. A history of intestinal trouble of doubtful nature, though most probably dysenteric was obtained in six cases. In the remaining 10 cases, previous dysentery was denied and yet the clinical manifestations of amoebic hepatitis were present in all but one. However this is not an uncommon feature, Manson Bahr, in an analysis of 50 cases of amoebic liver abscess, reported that he could get history of previous dysentery in 30 cases and could discover amoeba or cysts in stools in only 23.

The presenting chest symptoms were the following, arranged in order of frequency:

	No. of Cases	Percentage
1—Pain	24	86
2—Hoemoptysis	17	61
3—Cough with mucopurulent sputum	11	39
4—Dyspnea	3	11

Pain was by far, the commonest presenting symptom. It is of a stitching character, localised in most cases to the right lower intercostal spaces either in the axillary or in the inframammary zones, sometimes referred to the right shoulder and in many cases associated with right hypochondriac dull aching pain.

It was a manifest symptom in 24 cases (86 per cent).

Next in frequency was hoemoptysis which was met in 17 cases. Its amount varied between just spitting of blood in seven and recurrent profuse bleeding in ten cases amounting to about half a litre each time, in some of them. Again the character of blood was variable. It was not necessarily of the peculiar types described as anchovy sauce coloured by some authors and as chocolate coloured by others. In our cases it was bright red blood in many cases. It is noticeable that most of the lung abscess cases were characterised by profuse hoemoptysis whereas the incidence of profuse hoemoptysis in the pneumonitis cases was equal to that of blood tinged sputum.

Cough accompanied with mucopurulent sputum little or abundant but no hoemoptysis, was the complaint of 11 patients. In four of them, it was a main symptom but in seven it was trivial and it was noticed that these latter cases were the ones that mainly manifested by pleural involvement whether it was a serous pleurisy secondary to the underlying liver trouble or empyema caused by rupture of an amoebic liver abscess through the diaphragm into the pleura.

Table I illustrates the possible influence of the chest lesion on the type of cough and sputum the patient had. Dyspnea was a common toxic manifestation in many cases but the main presenting symptom in the 3 empyema cases.

At last and apart from the above mentioned presenting chest symptoms we had one case that presented as an acute involvement of the abdomen but clinical examination revealed the presence of an enlarged tender liver and a small right pleural effusion. The case, when admitted to the medical ward, proved to be one of amoebic hepatitis with complicating pleurisy and was cured by emetine.

Physical Signs

Evidence of toxoemia in the form of low grade fever (37.5-38°c) appeared in 19 cases (64 per cent) and clubbing of the fingers in 17 cases (61 per cent). In one clubbing was of an advanced degree, the so-called drum stick clubbing but in the remaining 16 it was moderate.

Examination of the abdomen revealed an enlarged tender liver in 22 cases (80 per cent) due to amoebic hepatitis or amoebic liver abscess. Enlargement measured two to five finger-breadths and in two there was an overlying oedema of the skin. In the remaining six, the liver was clinically normal.

Table II illustrates the relation between the type of chest lesion and the condition of the liver, considering the presence or absence of previous history of dysentery.

It is remarkable that history of previous dysentery was present or at least doubtful in almost all cases where the lung was involved by amoebic lung abscess, pneumonitis or bronchitis whereas of the 10 patients who denied previous dysenteric symptoms, five had no involvement of the lung parenchyma or the bronchi and they only had serous pleural effusion complicating an underlying amoebic disease of the liver. Could that suggest the possibility of a difference that exists in the severity of intestinal infection which is reflected on the ability of amoeba to metastasise in the lungs? or is it a coincidence? Another observation is that we noticed that no

TABLE I
CHARACTER OF COUGH AND SPUTUM IN RELATION TO THE TYPE OF CHEST LESION

Chest Lesion	Total Number	Profuse Hoemoptysis	Blood Tinged Sputum	Abundant Mucopurul. Sputum	Trivial Cough and Sputum
All cases	28	10	7	4	7
a—Amoebic lung abscess	9	6	2	1	....
b—Amoebic pneumonitis	9	4	4	1	....
c—Amoebic bronchitis	2	....	1	1	....
d—Serous Pl. Eff.	5	....	....	1	4
e—Empyema	3	....	....	....	3

TABLE II
AFFECTION OF THE CHEST IN RELATION TO CONDITION OF LIVER AND HISTORY OF DYSENTERY

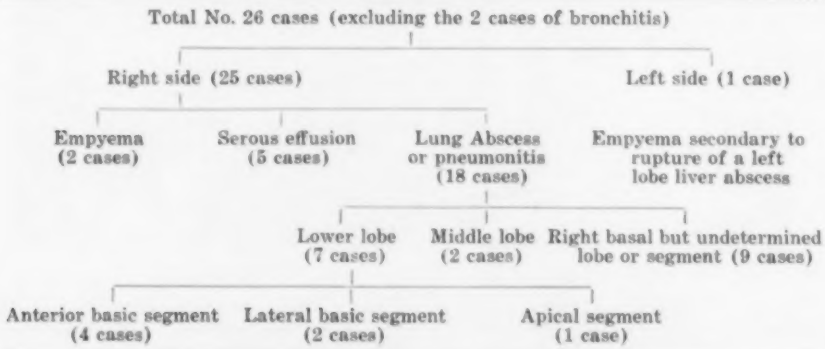
Chest Lesion	Total Number	History of Dysentery: Present	History of Dysentery: Absent	History of Dysentery: Intestinal Trouble and Dysentery	Clinical Condition of Liver: Normal	Clinical Condition of Liver: Amoebic Hepatitis	Clinical Condition of Liver: Amoebic Liver Abscess
All cases	28	12	10	6	6	14	8
Amoebic lung abscess	9	6	1	2	4	5	....
Amoebic pneumonitis	9	4	2	3	2	4	3
Amoebic bronchitis	2	2	....	....	....	1	1
Serous Pl. Eff.	5	....	5	....	....	4	1
Empyema	3	....	2	1	....	....	3

case of amoebic liver abscess was complicated by an amoebic lung abscess. Could it be that a liver abscess might be present but cannot be diagnosed clinically whereas it can be discovered on pathological examination? Of course this is possible because a controversy has been demonstrable between the clinical and the pathological findings on many occasions and multiple abscesses were found in the livers in autopsies reported to have had normal livers clinically. The possibility of leakage of the liver abscess through the diaphragm into the lung has been described by Roger and we have already referred to it. It results in a marked diminution in the size of the liver abscess cavity which may shrink to the size of a finger tip, an event which will render it undetectable on clinical examination.

Localisation of the Lung Lesion

If we exclude the two cases of bronchitis, in the remaining 26 cases the lesion was situated in the right base in all of them except one that manifested as a left empyema caused by direct rupture of an amoebic ab-

TABLE III
LOCALISATION OF LUNG LESION



scess of the left lobe of the liver through the left diaphragm into the pleural sac. This was a butcher aged 30 who presented to us with dyspnea, low grade fever and stitching pain in the left axillary region. Clinical examination revealed the presence of left pleural effusion and a soft, tender mass in the epigastrium encroaching over the left hypochondrium. It was the chocolate coloured pus obtained on exploration of that pleural effusion that suggested to us the possibility of its amoebic nature. Again failure of the usual lines of treatment including aspirations and local instillation with penicillin and streptomycin in contrast to the dramatic response of both abdominal and chest manifestations to emetine treatment helped to assure our diagnosis. Figure 1 illustrates an example of middle lobe localisation.

This man of 22 years presented mainly with recurrent hoemoptysis for six months. It was the chocolate colour of sputum which attracted our attention in this case. However, penicillin was tried at first, having failed to detect tubercle bacilli in sputum repeatedly. When no improvement was achieved, a dramatic result was obtained by 6 grains of emetine. But in this case, a second course of 10 grains of emetine was needed one month later because of a persistent residual patch of pneumonitis and he was cured. Figure 2 demonstrates an example of localisation in the anterior basic segment of the right lower lobe. In this case hoemoptysis of about half a litre of bright red blood recurred at intervals over a period of three months. There was a history of untreated amoebic dysentery and E. histolytica cysts were detected in stools. Again in this case, response was only obtained and was dramatic to emetine treatment.

Pleural Manifestations

The pleura was involved in 14 cases, all of which were on the right side except the one mentioned above.

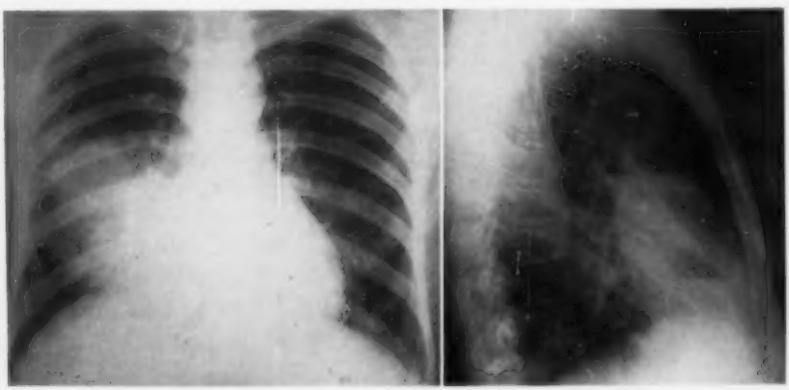


FIGURE 1A FIGURE 1B

Figure 1A: Postero-anterior view of the chest of a case of middle lobe amoebic lung abscess.—*Figure 1B:* Right lateral view of the same case, confirming the middle lobe localisation of the lesion.

(1) Pleurisy with serous effusion: 11 cases

A. as the only chest manifestation in five cases.
   i. in three it was complicating amoebic liver abscess,
   ii. in two it was complicating amoebic hepatitis.

B. Associated with other chest manifestations in six.
   i. in five associated with right amoebic pneumonitis and complicating amoebic hepatitis,
   ii. in one associated with amoebic bronchitis and complicating an amoebic liver abscess.

(2) Empyema: three cases. In each, there was a big collection of chocolate coloured pus in the pleural space on the right side in two and on the left side in the third. In all three cases, culture of the fluid for various organisms and amoeba was negative and failed to reveal any.

It is evident that empyema is caused by rupture of liver abscess, through the diaphragm into the pleural sac. This is confirmed by the character of the fluid, being sterile on culture and the dramatic response to emetine.

In one case emetine was given intrapleurally in addition to the usual intramuscular course. In this case the patient had a history of epigastric discomfort and mild fever followed by stitching pain in the right axillary region and shoulder with cough and anchovy sauce coloured sputum and he soon developed severe dyspnea. The liver was enlarged three finger-breadths and tender and massive right chocolate coloured pleural effusion was present. Preliminary aspirations and treatment with penicillin and streptomycin were of no benefit. Then emetine one grain daily was started and the first aspiration of 500 cc. was followed by one quarter grain of emetine intrapleurally. Three days later one-half grain and three days later one grain of emetine intrapleurally followed aspiration. No more fluid could be obtained on puncture and the patient was cured but we advised an additional course of Aralen. The great success in treating this case with

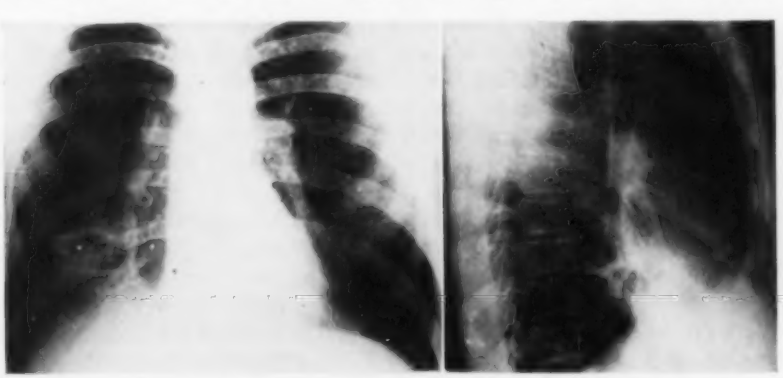


FIGURE 2A FIGURE 2B

Figure 2A: Postero-anterior view of the chest of a case of right lower lobe amoebic lung abscess.—*Figure 2B:* Right lateral view of the same case, showing localisation of the lesion to the anterior basic segment.

emetine can be appreciated if compared to the result of its previous treatment with antibiotics.

As to the other cases where pleurisy was accompanied by serous effusion, we are not certain of etiology.

Laboratory Findings

Blood picture: Significant changes were only detected in the leukocyte count which varied between 5,600 and 23,400. Apparently this was not affected by the lung lesion but related to the liver condition, for instance:

In three liver abscess cases, the total leukocyte count was more than 20,000. In four (three of liver abscess and one of amoebic hepatitis) the total count varied between 15,000 and 20,000. In eight cases it varied between 10,000 and 15,000 and in 13 it was less than 10,000.

In the differential picture, noticeable changes were observed in the polymorphonuclears and in the eosinophils. The lowest polynuclear count was 48 per cent of 6,000 total, i.e., 2,880 and the highest was 91 per cent of 23,400 total, i.e., 21,294 polymorphs. In the remaining 26 cases the total polynuclear count varied between 4,960 and 8,160.

The eosinophil count was 0 to 6 per cent in 20 cases, 8 per cent of 8,400 in one case and 14 per cent of 6,400 in another. The average total number of eosinophils was 400 and the highest was 1,200.

Search for amoeba in its vegetative form or as cysts:

1) Stools: examination in all cases was positive in six.
2) Pus from liver was negative in three cases.
3) Sputum of nine was negative.
4) Bronchoscopic specimens obtain in five cases were all negative.
5) Pleural fluid in the three empyema cases revealed no amoeba.

These results agree with almost all reports in the literature to the effect that amoeba can rarely be detected in the sputum.

Diagnosis

In most cases this was based on evidences obtained from the history, the right basal localisation of the lung lesions, the character of the sputum and the associated liver condition in presence of laboratory findings that could exclude other possibilities. Diagnosis is more assured by failure of other specific remedies and the dramatic responses to emetine.

Difficulties were met in differentiating them from pulmonary tuberculosis and pneumonias including the suppurative forms. The long observation without specific treatment or under drugs other than antiamoebic ones without improvement, compared with the rapid and dramatic improvement that progressed to complete cure once antiamoebic treatment was started should clarify etiological diagnosis.

We have two cases where histories and clinical picture favoured the diagnosis of amoebic bronchitis and more support was offered by the dramatic response and complete resolution that rapidly followed antiamoebic treatment.

In a third case, the suggestion of amoebic bronchitis was considered in retrospect.

At any rate, amoebic bronchitis without abscess of lung has been described clinically by various authors but the condition has not been encountered at autopsy. Petzetakis describes amoebic bronchitis occurring in some cases as the only manifestation of amoebiasis caused by metastasis of amoeba from a latent focus in the intestine or liver or by inhalation of dust containing amoeba.

Treatment

Emetine hydrochloride was used in all cases with remarkable success, so much so, that it is justifiable in doubtful cases as a therapeutic test. The general belief is that it is a specific drug and under experimental conditions destroys E. histolytica in a dilution of 1: million though Dobell showed that it is lethal to amoeba in culture in a strength of 1:5 millions. It is reported to be more effective in metastatic lesions than in intestinal infection and Manson Bahr advises the following as a full course:

One grain daily for seven days, then two grains daily for four days and then one-half grain daily for three days.

In spite of the fact that the specific action of emetine against amoebic infection is accepted beyond doubt we were interested to learn whether it can exert any lethal action on other organisms. Dr. S. El-Dewi of the clinical pathology department examined sputum from 10 cases of ordinary pyogenic lung abscesses and the following were the types of strains isolated on culture:

10 strains of pneumococcus,
5 strains of N. catarrhalis,
4 strains of strept. viridans,
3 strains of Klebsiella,
1 strain of N. flavus, and
6 strains of diphtheroids.

He tested these strains for sensitivity to emetine hydrochloride by the cup method. The dose used was 1:million dilution which is several times the dose used by Godwin et al., (1948) and Anderson (1950). In all instances the organisms were found resistant to the drug and growth proceeded to the edge of the cup. So, evidently these laboratory findings confirm the clinical observations that no organism is affected by emetine except the E. histolytica.

The second observation is that no case of this series was benefited by any other chemotherapeutic or antibiotic given and this is in full agreement with what has been reported by McHardy and Frye who evaluated the following antibiotics as amboebicides in 1,550 cases: chlorotetracycline (Aureomycin), oxytetracycline (Terramycin), fumagillin, chloramphenicol and carbomycin. Studies showed that oxytetracycline was the most effective antibiotic of that group for treatment of intestinal amoebiasis and the authors state that hepatitis, hepatic abscess and other extracolonic amoebic involvements are not benefited by treatment with the antibiotics evaluated.

We think emetine is preferable to chloroquine and we believe it is the opinion of other workers too, except Chakravarti who reported that he obtained an earlier and a more complete clinical response with chloroquine than with emetine.

In this series 15 cases were given one course of 6-10 grains of emetine, two received one course of 12 grains, three were given emetine followed by chloroquine (Aralen), and eight received two courses of emetine, a first of 6 grains and another of 10 grains following a rest period of one to

FIGURE 3A

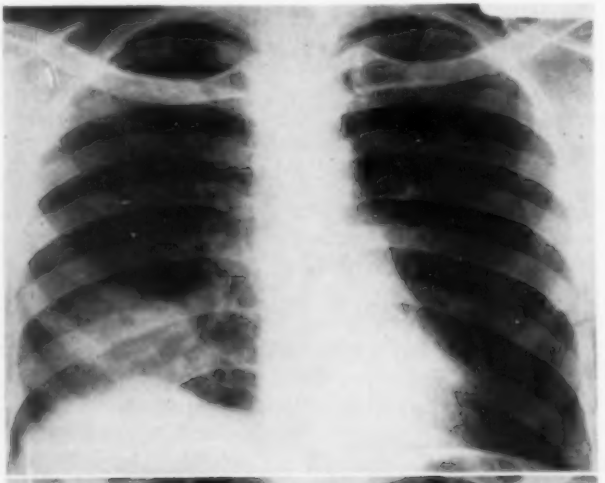


FIGURE 3B

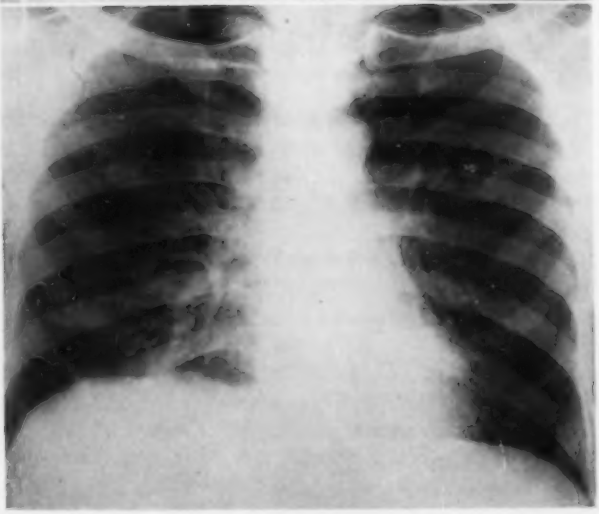


Figure 3A: Postero-anterior view of the chest of a case of right basal amoebic lung abscess before treatment.—*Figure 3B:* Postero-anterior view of the same case after treatment with emetine.

four months usually because of the occurrence of a relapse or because of the persistence of some suspicious residual shadow.

In one of the cases, emetine was given both parenterally and inside the hepatic abscess cavity and in another it was given intrapleurally in addition to the usual parenteral course.

Result of Treatment

There was dramatic success in all cases, an example of which is illustrated in Figure 3. We followed our cases for more than one year with the following end results:

	Number	
1—Complete cure	19 cases	
2—Residual radiographic shadow caused by:		
a—pleural thickening	3 cases	5 cases
b—residual fibrosis (normal bronchography)	2 cases	
3—Bronchiectasis proved by bronchography	2 cases	
4—Segmental atelectasis (radiological observation)	2 cases	

SUMMARY

A detailed analysis is given of a series of 28 cases with bronchopulmonary manifestations of amoebiasis.

The series includes nine cases of pneumonitis, nine lung abscess, two bronchitis, four pleurisy with serous effusion and three empyema.

Twenty two were secondary to spread from associated amoebic hepatitis or amoebic liver abscess and six were not accompanied by clinical evidence of disease in the liver and hence considered as examples of primary pulmonary amoebiasis.

The commonest clinical manifestation of bronchopulmonary amoebiasis was pneumonitis or lung abscess of the lower zone of the right lung; hoemoptysis and localised chest pain were the most frequent presenting symptoms.

Diagnosis was based on evidences obtained from history, right basal localisation of the lung lesion, character of sputum and associated liver condition and it was assured by the dramatic response to emetine hydrochloride.

Working in an endemic locality, the physician should be amoebiasis conscious, when dealing with any right basal pneumonic lesion particularly if it is of a suppurative nature.

RESUMEN

Se presenta un detallado análisis sobre una serie de 28 casos con manifestaciones broncopulmonares de amibiasis.

Las series incluyen nueve casos de neumonitis, nueve de abscesos pulmonares, dos de bronquitis, cinco pleuresías con derrame seroso, y tres empiemas. Veintidos fueron secundarios a invesión a patrir de hepatitis amibiana o absceso amibiano hepático y seis no se acompañaron por evidencia clínica de enfermedad del hígado y por tanto se consideran como ejemplos de amibiasis pulmonar primaria.

Las manifestaciones clínicas de amibiasis broncopulmonar fueron neumonitis o absceso pulmonar de la base derecha; hemoptisis y dolor localizado fueron los síntomas más frecuentes.

El diagnóstico se basó en la evidencia proporcionada por la historia clínica. Localización basal derecha, carácter del esputo y afección asociada del hígado así como la espectacular respuesta a la emetina.

El médico que trabaja en una área donde la amibiasis es endémica debe tenerla presente cuando se trate de una lesión neumónica basal derecha especialmente si es de naturaleza supurativa.

ZUSAMMENFASSUNG

Es wird eine ins Einzelne gehende Analyse gegeben von einer Reihe von 28 Fällen bronchopulmonalen Manifestationen der Amöbenruhr.

Die Reihe umfasst neun Fälle Pneumonitis, neun mit Lungenabszess, zwei mit Bronchitis, fünf mit Pleuritis und serösem Erguss und drei mit Empyem. 22 Kranke waren sekundäre Formen mit Ausbreitung von einer gleichzeitigen Amöben - Hepatitis oder Amöben - Leberabszess und sechs Patienten waren nicht vergesellschaftet mit klinisch evidenter Leberer-krankung und werden daher angesehen als Beispiele primärer pulmonaler Amöbenruhr. Die häufigste klinische Manifestation der bronchopulmonalen Amöbenruhr war Pneumonitis oder Lungenabszess im unteren Abschnitt der rechten Lunge; Haemoptyse und lokalisierter Brustschmerz waren die am häufigsten vorliegenden Symptome. Die Diagnose gründete sich auf den Befund anhand der Vorgeschichte. Lokalisation des Lungenbefundes rechts basal, Charakter des Sputums und zugehörige Leberbeschaffenheit und die Diagnose wurde bestätigt durch die dramatische Reaktion auf Emetin-Hydrochlorid.

Der in einem endemischen Bezirk tätige Arzt muss an Amöbenruhr denken, wenn er es mit einer rechtsseitigen basalen Lungenveränderung zu tun hat, besonders wenn sie von eitriger Natur ist.

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SECTION ON CARDIOVASCULAR DISEASES

The Use of a Myocardial Electrode Inserted Percutaneously for Control of Complete Atrioventricular Block by an Artificial Pacemaker*,**

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The occurrence and persistence of complete heart block has constituted an important source of morbidity and mortality for patients undergoing open heart surgery for repair of congenital septal defects.

Intensive efforts to improve the management of this complication have been made by our surgical group[1-4] and this knowledge has contributed to a significant reduction in mortality. The placement of a myocardial electrode[2-4] during surgery has proved to be the best method for control of heart block present following operative procedures. Very occasionally, however, complete and persistent heart block has not occurred at the time of surgery but has had a late onset in the days following the operative procedure and at that time reliance had to be placed upon drug (Isoproterenol† U.S.P.) therapy inasmuch as this complication has not occurred frequently enough to make it practical, in our opinion, to insert a myocardial wire prophylactically as a routine procedure. However, it should be mentioned that in selected patients‡ where there has been reason to suspect the likelihood of atrioventricular dissociation occurring again subsequently, we have implanted a myocardial electrode prophylactically since no deleterious effects have been recognized experimentally or clinically from the temporary presence of this fine wire in the right ventricular musculature.

Moreover, since the myocardial electrode has been so effective in controlling the heart rate of surgical patients with complete atrioventricular dissociation, it has seemed logical to consider methods for application of

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†Isuprel, Winthrop Laboratories, New York City.
‡Those patients with temporary episodes of complete block at time of surgery, or unusual irregularities of the heart rate observed immediately post-cardiotomy have had a myocardial electrode inserted prophylactically before closing the thoracotomy.

comparable life-saving benefits to non-surgical patients sustaining atrioventricular dissociation of an ominous degree as a complication of myocardial infarction, drug therapy, or one of the other diverse causes of Stokes-Adams syndrome.

Therefore, a method using a fine wire introduced percutaneously into the myocardium has been developed experimentally and has appeared effective and feasible for clinical usage.

Method of Study

Fifteen dogs weighing 14 to 23 kilograms were used for these experiments. Creation of complete heart block, trials of percutaneous myocardial insertion of wires and tests of the effectiveness of the control of heart block by an electrical pacemaker connected to these internal electrodes of various types have constituted the methods of study.

Procedure for Experimental Production of Atrioventricular Block:

The animals were anesthetized with 2.5 per cent intravenous pentothal sodium after having been premedicated with morphine and atropine and were ventilated with a positive pressure respirator connected to an endotracheal cannula during the operative procedure. A right thoracotomy was performed in the bed of the fifth rib or in the fourth intercostal space. The pump-oxygenator used for cardiopulmonary bypass was a plastic sheet oxygenator*[5, 6] in conjunction with a Sigmamotor pump,** type 6-S. The cannulation of the superior and inferior venae cavae was performed through the azygos vein and the auricular appendage respectively. Umbilical tapes around the venae cavae tightened through rubber cuffs served to divert the venous blood into the pump oxygenator. The arterial catheter was placed in the common femoral artery. Heparin (1.5 milligram per kilogram body weight) was given intravenously just prior to the cannulation and protamine sulfate (3 milligrams per kilogram body weight) was administered at the conclusion of the bypass. The flow rate was about 40 to 50 cc. per kilogram body weight per minute.

Through a right atriotomy, the area of the atrioventricular (A.V.) node and bundle was located and one or more stitches placed to interrupt its continuity as previously described.[4] In canine hearts, the atrioventricular node is usually located in the lower portion of the interatrial septum just above the annulus along a diagonal line between the ostium of the coronary sinus and the membranous septum. The atrioventricular bundle proceeds horizontally and cephalad reaching the inferior edge of the membranous portion of the ventricular septum where it divides into right and left bundle branches. This pattern may be demonstrated visually in the very fresh heart by painting the septum with Lugol's solution.

Electrocardiograms were recorded during all experiments to correlate the effects of these stitches with the observations upon the ventricular rate. Sometimes the achievement of complete atrioventricular block has

*Travenol Division of Baxter Laboratories, Inc., Morton Grove, Illinois
**Sigmamotor, Inc., Middleport, New York.

required the placement of two or occasionally three stitches. The insertion of these stitches should not be too deep or too far anterior lest the aortic cusps (they were involved two times in the beginning of these experiments) be injured.

In five animals complete atrioventricular dissociation was observed to disappear after the closure of the atriotomy (transient complete atrioventricular block). Additional cardiotomies were performed (ventriculotomy three times, new atriotomy two times) in those animals in order to place supplementary sutures around the atrioventricular bundle before production of permanent block was achieved. In three of these dogs, incisions in the area of the atrioventricular node and bundle were used in conjunction with stitches. By these means a complete and permanent atrioventricular block was created in all animals. After closure of the atriotomy and removal of the cannulae, chest catheters were placed in the hemithorax and the thoracotomy incision closed.

Technique for Percutaneous Insertion of a Myocardial Wire Experimental Studies:

A needle was inserted through the sixth right intercostal space near the sternum. A wire was pushed through the needle and advanced into the myocardium of the right ventricle five millimeters beyond the point at which the cardiac impulse was first felt. The outer needle was then removed and the wire which was left in place was connected to the pacemaker. A subcutaneous needle placed on the left lateral chest wall near the cardiac apex served as the indifferent electrode. The threshold for effective stimulation was determined by gradually increasing the current amplitude from 0 until effective stimulation of the ventricular beat resulted. Effective stimulation over prolonged intervals was maintained at a current amplitude a little above these threshold levels. The frequency was set at 90 to 110 impulses per minute. ECG tracings were recorded in different leads and the peripheral pulse measured. Pacemaker stimulation was maintained for six hours in these animals and the examinations repeated every half hour to assess the efficacy of the cardiac stimulation. At times the electric stimuli were varied in frequency and interrupted momentarily in order to demonstrate the dependence of the ventricular responses upon them. In one dog, two myocardial electrodes inserted percutaneously were used without indifferent electrodes and stimulation by a transistorized pacemaker maintained successfully for ten days.

Results:

Several kinds of wires were tested experimentally as myocardial electrodes. In the beginning, a semi-rigid wire insulated by passage through polyethylene tubing was used successfully, but its relatively large caliber (1 millimeter), requiring a large needle, and its lack of flexibility led to its abandonment. A stainless steel braided surgical wire (Surgaloy), size 0, insulated by passage through fine polyethylene tubing was tested next. This worked well and it was also tested without insulation which facilitated introduction through the lumbar puncture needle (No. 20). The stimula-

tion was found to be equally successful with or without the insulation (Figure 2). Thus, this size wire without insulation was used for the last eight experimental studies and possesses the qualities deemed practicable for clinical usage.

The myocardial electrode inserted percutaneously has controlled complete atrioventricular block in every instance, producing effective ventricular contractions at the desired rate as demonstrated by ECG response and femoral pulses synchronous with each electrical impulse. The threshold for effective cardiac stimulation ranged from 2.5 to 4.0 volts. When the rates of stimulation were slower than the intrinsic cardiac rhythm, intermittent ectopic beats appeared. With rates of stimulation faster than the intrinsic ventricular rate the stimuli provided complete control of the ventricular rhythm and regular pulses.

In some cases, the chest was opened to inspect the site of insertion of the electrode into the myocardium and no bleeding points were visualized. Postmortem examination of dogs showed no damage from the wire or the electric current to the myocardium and to neighboring structures. The point of puncture was scarcely visible and examination of the right ventricular cavity showed no damage to the endocardium.

Effect of Isuprel Upon the Heart:

In three animals, the effect of isoproterenol hydrochloride (U.S.P.), Isuprel®*, was studied. An intravenous injection of 2 cc. (1-50,000) of

*Winthrop Laboratories, New York City.

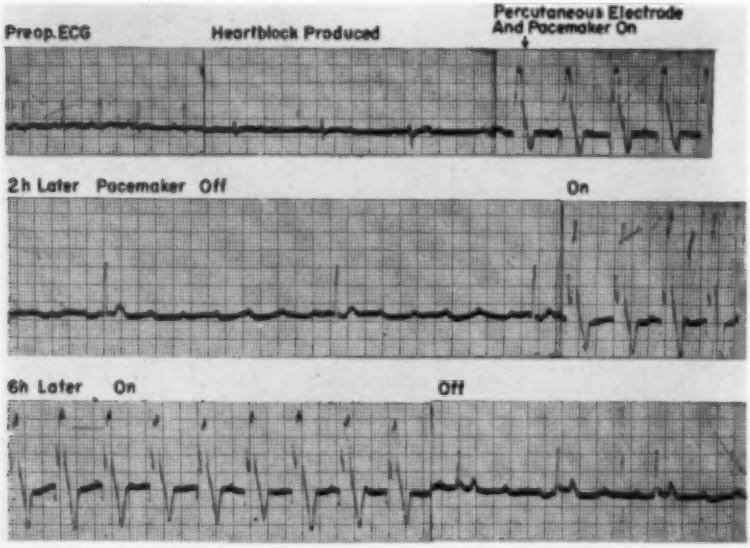

FIGURE 1: Complete atrioventricular dissociation produced experimentally and its control by an uninsulated myocardial (right ventricle) electrode inserted percutaneously (Dog. No. 100).

Isuprel produced effective ventricular contractions, but of a short duration lasting 3 to 10 minutes (Figure 3). Subsequent intravenous injections or subcutaneous implantation of tablets showed an improvement in cardiac rate but without complete control. These studies are further confirmation of the experimental[7] and clinical observations[1-4] upon the effect of Isuprel in temporarily increasing cardiac rhythmicity and rate.

Summary of Experimental Studies:

The percutaneous insertion of an uninsulated wire into the myocardium through a needle has proved to be a satisfactory, safe, and easily applicable method of controlling the slow cardiac rate and diminished cardiac output resulting from complete heart block created surgically. This method of control has important advantages in terms of effectiveness over the external electric pacemaker or use of the drug Isuprel.

Investigational Studies Upon a Technique of Percutaneous Insertion of the Myocardial Electrode in Man

The human technique to be described was developed mainly from studies carried out in cadavers. Seven patients who died and in whom autopsies were scheduled were used for this portion of the studies, 3 adults (2 females, 1 male, between 40 and 60 years), 1 female adolescent (16 years), 2 children (1 male—6 years, 1 female—4 years) and 1 infant (4 months).

The No. 0 braided stainless steel wire (Surgaloy) was introduced percutaneously into the myocardium of the right ventricle through a No. 20 needle and after removal of the anterior chest wall the site of myocardial insertion was checked. The right ventricular chamber was explored by a finger introduced into the pulmonary artery to feel the tip of the wire and its location before opening the heart and examining the endocardial surface. On each occasion the point of the myocardial insertion was found at about

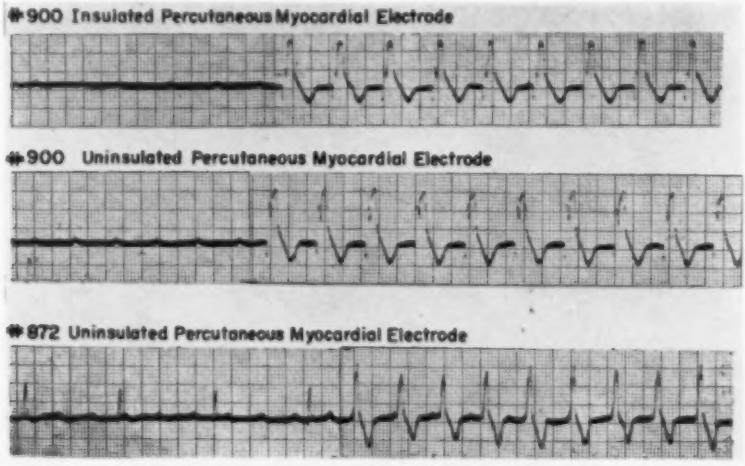

FIGURE 2: Comparative effectiveness of the control of complete block by insulated and uninsulated myocardial electrodes inserted into the right ventricle percutaneously. In Dog 900 the indicated comparison was made with identical pacemaker currents.

the same level on the anterior right ventricle wall. This was in the free area between the right coronary artery and the anterior descending branch of the left coronary artery about 2 cm. from the latter and about 2 to 3 cm. above the inferior margin of the right ventricle. In adults with a short and broad thorax the cutaneous mark for puncture was the fourth left intercostal space, and in those with long and narrow thorax the fifth left intercostal space at about 6 to 7 cm. from the midsternal line is recommended. In children, it was the fifth left intercostal space, 4 to 5 cm. from the midsternal line. The direction given to the needle is indicated below. A chest roentgenogram was taken in one instance to demonstrate the position.

In each instance the pleura was avoided as well as the parietal vessels and coronary arteries. No damage was observed on the myocardium or the endocardium. Passage of the tip of the wire into the ventricular cavity was noted on three occasions and does not appear to be of significance. The stimulation is provided by the contact of the wire with the ventricular myocardium.

Clinical Procedure (Figure 4) :

The only equipment needed is a lumbar puncture needle (No. 20 gauge) 3½" in length and a surgical stainless steel wire, size 0. The patient lies supine with an electrocardiograph connected. An electric pacemaker which has been converted to the low voltages (0 to 7.5 volts) needed for the internal electrode stimulation should be available for use.

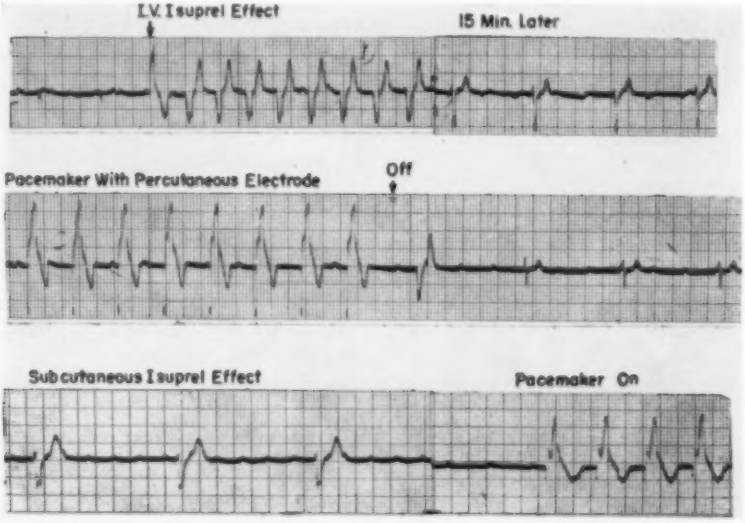

FIGURE 3: Electrocardiographic tracings depicting comparative effects of Isuprel and myocardial electric stimulation by a percutaneous electrode upon ventricular rate in a dog with complete block.

The effects of Isuprel upon cardiac rhythmicity are pronounced but evanescent, whereas, the ventricular rate remains completely responsive to repietive electrical stimuli of small amplitude.

The fifth left interspace and second right costo-chondral junction are located on the chest wall. A previous chest x-ray (if available) showing the exact size and configuration of the heart allows one to choose the site for puncture very accurately. After cutaneous infiltration with local anesthetic solution, the needle with the wire inside is inserted in the fifth left intercostal space at 4 to 5 cm. from the midsternal line (usually in children). The needle is then directed medially, dorsally, and cephalad towards the second right costo-chondral junction with an angle of 30° with the body surface (that is, pointed towards the back). Once the cardiac impulse is felt with the tip, the needle is advanced 5 mm. into the myocardium of the right ventricle. Then the wire is carefully advanced 1 cm. into the myocardium while the needle is withdrawn pushing in the wire a little so that the wire tip is not removed from the myocardium and also to provide a loop of wire to remain within the anterior mediastinum to guard against the removal of the wire by the heart beats. The wire is connected with the internal or negative electrode of the pacemaker. The electrical circuit necessary for ventricular stimulation is completed in one of two ways. A second percutaneous electrode may be inserted into the myocardium immediately adjacent to the first, or the circuit can be completed by placing a small flat indifferent electrode of stainless steel subcutaneously approximately over the site of the apical impulse beat. The threshold of effective stimulation is then determined by increasing the current amplitude from 0 until the ventricles respond at the desired rate.

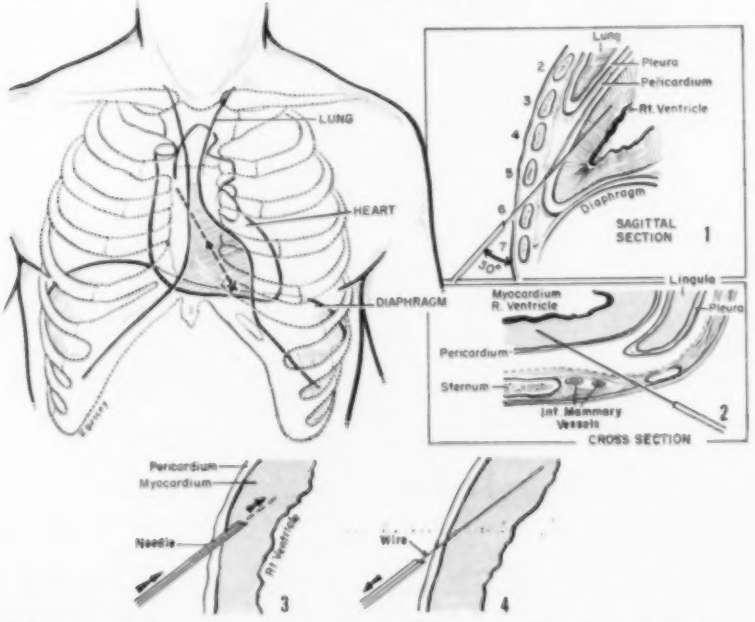

FIGURE 4: Technique for percutaneous insertion of a myocardial electrode (refer to text for detailed description).

An ECG tracing showing ventricular responses and the palpation of synchronous peripheral pulses are evidences of effective stimulation. Then a stitch secures the wire to the skin at the point of puncture to avoid removal due to movements.

The stimulation is maintained until the return of adequate rhythm and then is withdrawn easily. Although this remains to be fully evaluated by clinical trials, we see no insurmountable obstacles to keeping such electrodes in place for weeks or months if necessary for patients in chronic block and with dangerous low heart rates. Moreover, we see no contraindications to repeated reinsertion of the wire into the myocardium should it work loose in such patients. It was with these long term medical as well as surgical applications in mind that we developed and have in routine clinical usage a transistor pacemaker no larger than the hearing aids of a few years ago.

In the 55 patients with complete heart block treated to date, we have been able to maintain effective stimulation via a myocardial electrode placed at the time of thoracotomy for as long as 57 days. Further research is in progress at present to determine better methods for effecting a more permanent connection to the heart muscle for patients with chronic block.

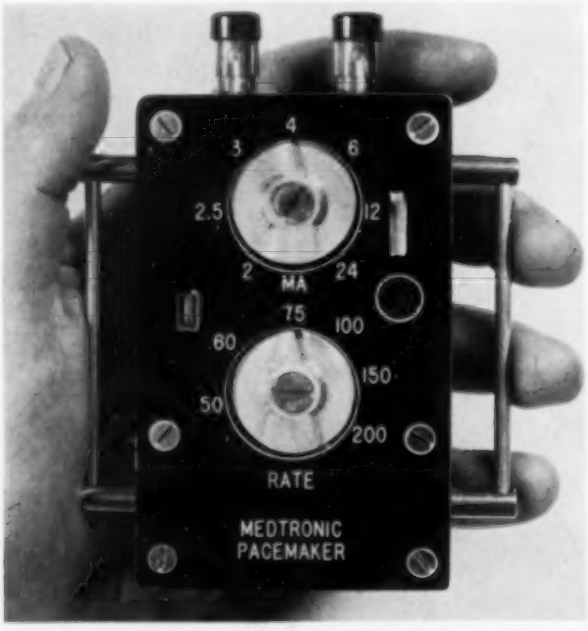

FIGURE 5: Featherweight transistorized cardiac pacemaker. The mercury battery is self contained.

This unit is designed for internal application only, where at least one electrode is attached directly to the ventricular myocardium. The instrument is small and light enough that it may be worn by the patient. The control knobs are recessed below the panel surface to prevent accidental changes. An output of 1 to 60 milliamperes (M.A.) is provided. A neon flasher visually indicates proper function of the pacemaker.

This need is more pertinent in non-surgical patients since the majority of surgical blocks revert to sinus rhythm within two to four weeks.

Should a thoracotomy incision provide a better means of establishing a permanent connection for these medical patients with a permanent block, we believe that the therapeutic gains would make such an operation entirely feasible because of the mortality and morbidity of the Adams-Stokes syndrome in those more seriously afflicted. Also under investigation in the animal laboratory is the use of an induction coil implanted within the cardiac muscle and activated from an externally placed coil without the need for wires through the chest wall.

Transistor Pacemaker:

In our earlier clinical experience with the myocardial electrode we utilized the standard commercially available pacemakers developed for external cardiac stimulation converted to give the low current amplitudes (1-60 milliamperes or 1 to 7.5 volts) desired. These instruments, while effective, have numerous disadvantages related to bulkiness, lack of portability, dependence upon external power sources, and inherent dangers to the patient should an internal short circuit occur.

Thus, a small (2.5 x 5 x 10 cm.) feather-weight transistorized pacemaker* with a self-contained battery sufficient for several months of continuous use has been developed and and used in 26 of these patients with the myocardial electrode. This pacemaker may be worn by the patient and has replaced completely the cumbersome units used earlier. Moreover, the battery unit is safer since power failures or electrocution due to short circuits are obviated.

Discussion

The purpose of the study was to develop a procedure for percutaneous insertion of a myocardial electrode to control persistent complete heart block occurring later in the postoperative course of cardiac surgical patients, and for use in non-surgical patients developing very slow ventricular rates with inadequate cardiac outputs as a complication of heart block due to cardiac disease or drug therapy.

The results of the experimental and clinical studies indicated that the complete atrioventricular block created by surgical injury of the atrioventricular conduction system can be successfully controlled by such a technique.

Evidence is presented herein that myocardial electrode inserted percutaneously and connected to a suitable electrical pacemaker assumes complete control of cardiac rhythm with a very low voltage and so eliminates the drawbacks of the external electric pacemaker with skin ulcerations, chest pain, and muscular twitch.

The technique described has proved to be effective, safe, and easily applicable. The percutaneous electrode need not be insulated.

*Medtronic, Inc., Minneapolis, Minnesota.

SUMMARY

1. A procedure to introduce percutaneously a myocardial electrode into the ventricular wall was developed.

2. Animal experimentation, with creation of complete and permanent atrioventricular block by placing stitches in the atrioventricular node and bundle through a right atriotomy under direct vision with the use of a pump-oxygenator, has shown the efficiency and the safety of this technique.

3. A technique for the percuntaneous insertion of the myocardial electrode in the humans is described. This method permits the effective control of complete heart block by use of the artificial pacemaker without the necessity of opening the chest to place an electrode.

4. The indications of the method are chiefly the control of complete heart block with very slow rates or episodes of ventricular standstill occurring for the first time in the postoperative course after the chest has been closed or in medical patients with the Stokes-Adams syndrome due to cardiac disease or drug therapy. The efficacy, safety, and easy applicability of this technique permits its use for emergency resuscitations.

5. The superiority of internal pacemaker stimulation over use of external electrodes or drug therapy (Isuprel) has been emphasized.

6. A lightweight transistor pacemaker has been developed which permits greater patient mobility and safety than the more cumbersome units available earlier.

RESUMEN

1. Se ha ideado un método para introducir un electrodo miocárdico por via percutánea.

2. Se ha mostrado la eficiéncia y la seguridad de ésta técnica en el animal de experimentación en el cual se ha creado quirúrgicamente un bloqueo completo y permanente de la conducción atrio-ventricular, colocando puntadas sobre el nodo atrio-ventricular y sobre el ház de His por atriotomía derecha bajo visión directa con la ayuda de la bomba oxigenadora.

3. Se describe una técnica para la inserción percutánea de un electrodo miocárdico en el hombre, lo que permite el control completo del bloqueo atrioventricular del corazón por medio de un marcador de paso artificial, sin necesidad de abrir el torax para la inserción del electrodo.

4. Las indicaciones del método son principalmente el control del bloqueo completo, con contracciones muy lentas o paro completo, que ocurre por primera véz en el postoperatorio de la cirugía cardíaca, después de haberse cerrado el torax, o en la resuscitación de los enfermos con el síndrome de Stokes-Adams, debido a enfermedad cardíaca o efecto de drogas. La eficacia, la seguridad y la aplicación fácil de ésta técnica permite su uso en resuscitaciones de emergencia.

5. Se ha demostrado la calidad superior de la estimulación del marcador de paso interior sobre los electrodos externos o el uso de drogas (Isuprel).

6. Un marcador de paso de poco peso con transistores se ha ideado, que permite un manejo mas facil y seguro del enfermo en comparación con los modelos anteriores mas grandes y mas estorbosos.

ZUSAMMENFASSUNG

1. Es wurde ein Vorgehen entwickelt, um eine Herzmuskel-Elektrode perkutan in den Herzmuskel einzuführen.

2. Eine tierexperimentelle Untersuchung mit Herstellung eines kompletten und permanenten A.V-Blocks durch Anwendung von Nähten am Atrioventrikularknoten and Schenkel durch rechtseitige Atriotomie unter direkter Sicht mit Benutzung eines Pumpen-Oxygenators, zeigte die Wirksamkeit und Sicherheit dieser Technik.

3. Die Technik zur perkutanen Insertion der Herzmuskelelektrode beim Menschen wird beschrieben. Dadurch wird es möglich, einem kompletten atrio-ventrikularen Herzblock zu bekämpfen unter Zuhilfenahme eines künstlichen Impulsgebers, ohne das Öffnen des Brustkorbes zu benötigen, um eine Elektrode einzuführen.

4. Die Indikationen der Methode sind hauptsächlich gegeben in der Bekämpfung des kompletten Herzblocks mit sehr langsamen Herzschlag oder kompletten Stillstehen, wie es zum ersten Mal im postoperativen Verlauf nach intrakardialer Chirurgie auftritt, nachdem der Brustkorb geschlossen wurde, oder auch in der Wiederbelebung von Kranken mit Adams-Stokes Syndrom, sekundär zu anderen Herzkrankheiten oder Arzneieffekten. Die Wirksamkeit, Sicherkeit und leichte Anwendbarkeit dieser Technik gestattet es, sie einzusetzen für Wiederbelebungen in Notfällen.

5. Die Überlegenheit der Internen Stimulation des Impulsgebers gegenüber der externen Elektrode oder Medikamenten (Isuprel) wurde hervorgehoben.

6. Es wurde ein Leicht-Gewicht-Transitoren-Impulsgeber entwickelt, der grössere Beweglichkeit und Sicherheit des Kranken gestattet im Vergleich zu den früheren plumperen Einheiten.

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Tularemic Pericarditis

Report of Two Cases and Review of Literature

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Pericarditis is a rare complication of tularemia. A review of the literature reveals 28 cases of tularemia involving the pericardium. Eleven of these are referred to by Drobinskij[1] in the Russian literature. Twenty-five of the cases were associated with pneumonia, pleurisy, or both of these complications. A report of pericarditis due to ulcero-glandular tularemia by Ljung[2] in 1955, and one of the cases under discussion are the only instances of "isolated pericarditis" due to tularemia without pneumonia or pleurisy reported in the literature. Drobinskij[1] refers to three similar cases in 1947 without further description.

Report of Cases

Case 1: A 37 year old white married salesman was seen on January 9, 1957, with the chief complaint of "severe crushing chest pain" of one to two hours duration. He was in excellent health until two weeks prior to admission when he went rabbit hunting. While dressing one of the rabbits, he noticed the liver was covered with "white spots" and he discarded the animal. Five days later, a small lesion developed on the dorsal aspect of the right hand. On the following day, he developed pain, swelling,

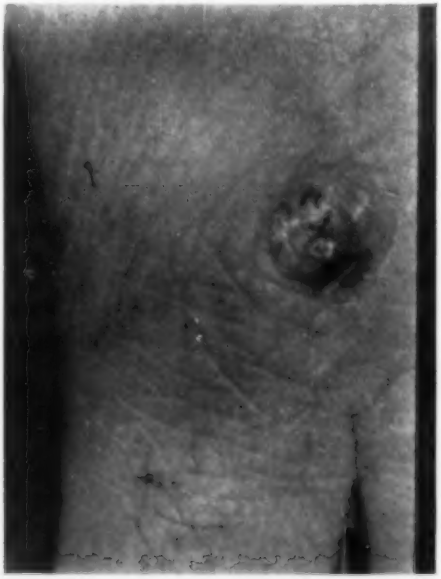

FIGURE 1: Note the small maculo-papular incrusted lesion 1 x 1 cm. on the dorsal aspect of the right hand.

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and tenderness in the right axilla. These symptoms were associated with fever, malaise, headache, and generalized muscular aching. The next day, he consulted his physician and was told he had "rabbit fever," and was treated with chloramphenicol, penicillin and streptomycin. He remained ambulatory and felt fairly well until two days later

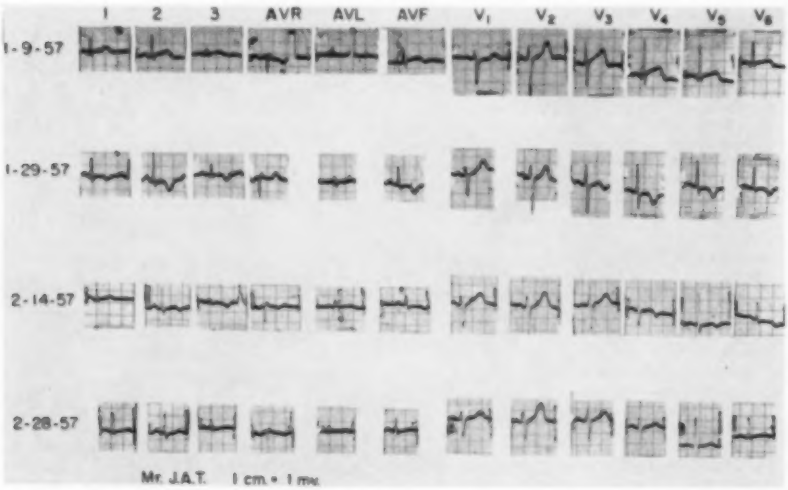


FIGURE 2: Serial electrocardiograms taken between January 9, 1957 and February 28, 1957 reveal the characteristic T-wave changes found in acute pericarditis. There is slight ST-segment depression in Lead 2, AVF, V-4, and V-5 on January 29, 1957. A premature ventricular beat is present in Lead 3 on February 14, 1957. On February 28, 1957 the electrocardiogram returns to normal.

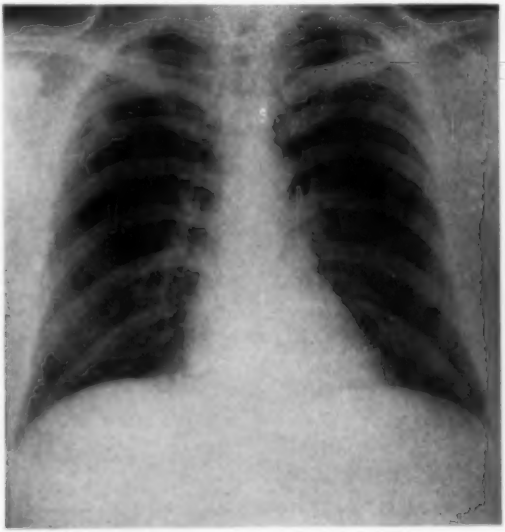


FIGURE 3: January 10, 1957. There is no cardiac enlargement. The lung fields are clear with the exception of a small calcified nodule in the right upper lobe measuring 1 cm. in diameter which is characteristic of a tuberculoma.

when he developed severe "crushing" substernal pain, associated with "shortness of breath." The pain lasted for one hour. On the following night the pain was more severe and lasted approximately two hours. In the morning because of severe, "crushing" substernal pain radiating to both arms, associated with dyspnea, shock, and cyanosis, the patient was admitted to St. Thomas hospital. After administration of 100 mg. of Demerol,® the patient's condition improved and signs of shock disappeared.

Physical examination revealed a temperature of 97.6°F. respirations 20 per minute and the blood pressure 100/70. There was a small maculo-papular incrusted lesion 1 x 1 cm. on the dorsal aspect of the right hand overlying the first metacarpal-phalangeal joint (Fig. 1). There was a tender, firm 1 x 2 cm. freely movable lymph node palpable in the right axilla. Chest was symmetrical with bilateral, equal expansion. Lung fields revealed no abnormal findings. Heart was not enlarged. There was a normal sinus rhythm with frequent premature ventricular contractions at a rate of approximate.y 70 per minute. Sounds were of poor quality and there was no friction rub. The remainder of the physical examination revealed no other abnormal findings.

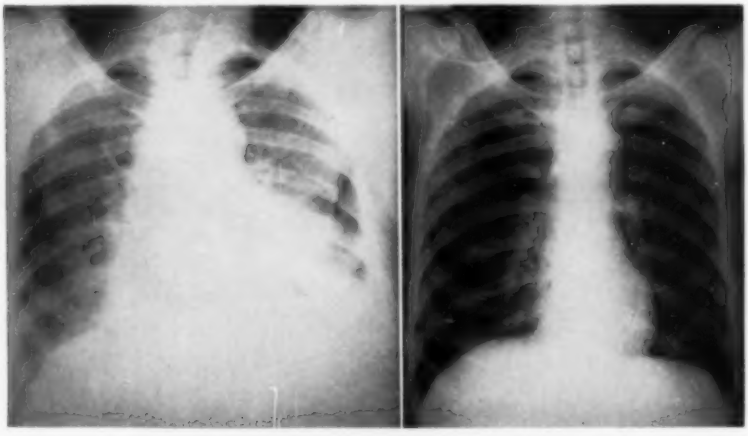
Serial electrocardiograms taken between January 9, and February 28, 1957 revealed electrocardiographic findings of acute pericarditis (Fig. 2). Chest roentgenogram January 10 showed no cardiac enlargement. There was a calcified nodule in the right upper lobe measuring 1 cm. in diameter, which was characteristic of a tuberculoma, otherwise the lung fields were clear (Fig. 3).

Laboratory: White blood cell count was 6,500 with a normal differential. The corrected sedimentation rate was 35 mm. Hemoglobin was 15.9 grams and the VDRL test was negative. Liver function tests were within normal limits and the C-reactive protein was not increased. The serum glutamic oxalacetic transaminase was 80 units. Urinalysis was negative.

The agglutination test for tularemia was negative on January 9, 1957, positive in a titer of 1:160 on January 28, 1:320 on January 30, and negative on February 14.

Course in hospital: On January 9, the patient was started on 0.5 Gm. each of chloramphenicol and streptomycin every six hours. On the following day, a pericardial systolic friction rub was audible and remained audible for one day. After three days in the hospital, he had no substernal pain but occasionally experienced a "dull ache" over the precordium. Frequent premature atrial and ventricular contractions were present throughout the hospital course. Axillary lymphadenopathy disappeared in seven days, and the temperature, pulse and respiration remained normal. On January 19, the doses of chloramphenicol and streptomycin were reduced to 12 hour intervals. The lesion on the right hand disappeared in three weeks. He was discharged in excellent condition on January 26, 1957.

Case 2: A 59 year old highway maintenance worker was seen in the medical clinic at Vanderbilt University Hospital with the chief complaint of "infected finger and



8-16-49 10-11-49

FIGURE 4: Note the marked enlargement of the cardiac silhouette with a small amount of plueral effusion, prominent lung markings, and pneumonitis at the left base on August 16, 1949 compared to the normal cardiac silhouette and clear lung fields on October 11, 1949.

pneumonia." Six weeks before admission he had "scratched" the fourth finger of the left hand on a nail. Two weeks later he developed fever, chills, weakness, with lymphangitis of the left arm and lymphadenitis in the left axilla. His local physician, treated him with sulfadiazine and penicillin without improvement. Weakness, anorexia, fever, and chills continued and he was given chlortetracycline for two days, which made him feel better. Two weeks later he developed a hacking non-productive cough, and the chest roentgenogram showed "pneumonia." Cough and weakness persisted, and the patient lost 28 pounds in weight. He became "short of breath" and developed "sharp pain" in the left upper chest region which was aggravated by coughing and deep breathing. There was no recollection of exposure to rodents, rabbits or other possible sources of infection.

On physical examination he appeared chronically ill with dyspnea and orthopnea. He was thin and apathetic with evidence of recent weight loss. Temperature 102°F, pulse 194, respirations 28 per minute, and the blood pressure 110/68. There was an incrusted, indurated 4 x 10 mm. lesion with circumscribed erythema on the fourth finger of the left hand. There was a tender, firm lymph node 3 cm. in diameter in the left axilla. Lung fields revealed absent tactile fremitus at the left base anteriorly and posteriorly. There were diminished breath sounds with moist rales extending from the left base to the inferior margin of the scapula, and crepitant rales were audible at the right base. Heart was enlarged 2 cm. to the left of the mid-clavicular line and to right 2 cm. beyond the lateral sternal margin. Heart sounds were distant and of poor quality with a normal sinus rhythm. Over the base, there was a to and fro friction rub. Radial pulses were paradoxical. No other physical abnormality was found.

Chest roentgenogram on August 16, 1949 demonstrated enlargement of the cardiac silhouette with a C-T ratio of 18 to 29 cm. There was a slight amount of fluid at the left base. Lung markings were prominent with pneumonitis present in the left base. A chest roentgenogram on October 11, revealed a marked decrease in the size of the heart. The C-T ratio was 11 to 28.5 cm., and the heart was long and linear. Lung fields were clear (Fig. 4). Serial electrocardiograms from August 11, showed changes characteristic of pericarditis (Fig. 5). Laboratory examination revealed normal urinalysis, white blood cell count 6,400 with normal differential, hematocrit 34 mm., and sedimentation rate (corrected) 32 mm. The VDRL test was negative. Agglutinations for tularemia; August 10, 1:320; August 16, 1:20,480; and on August 24, 1:320. Venus pressure and circulation time were normal.

Course in hospital: The patient improved promptly with 0.5 Gm. streptomycin every 12 hours but because of continued fever the dose of streptomycin was increased by

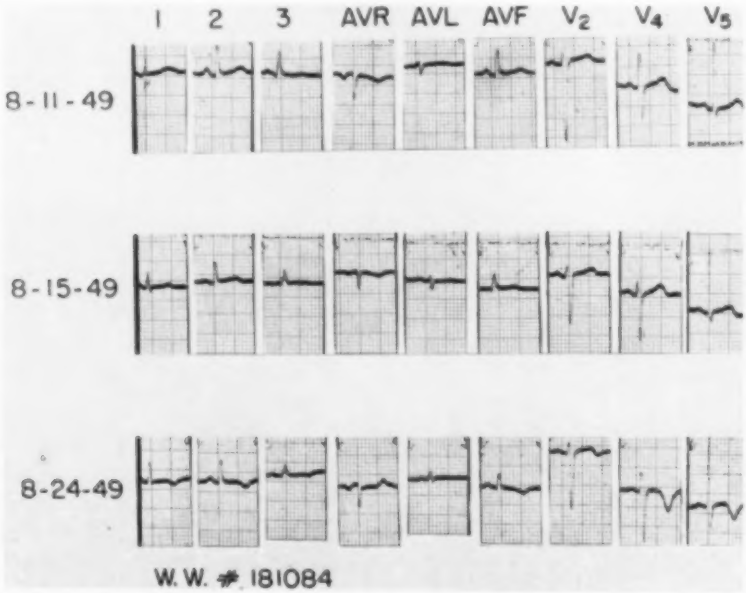


FIGURE 5: Serial electrocardiograms taken between August 11, 1949 and August 24, 1949 reveal T-wave inversion in all leads characteristic of acute pericarditis.

TABLE I—SUMMARY OF THE PULMONARY AND CARDIOVASCULAR MANIFESTATIONS OF TULAREMIC PERICARDITIS

Case Number and Author	Age Sex	External Lesion	Pneumonia	Pleural Effusion	Pericardial Rub	Other Signs of Pericarditis	Pericardial Tap	EKG Evidence of Pericarditis	Agglut.	Subsequent Course
1. Simpson 1929	37 Male	Yes	(?)	None	Yes	Precordial pain, dyspnea, x-ray and pericardial tap	Yes	No	1:60	Complete recovery
2. Pessin 1936	55 Female	None	Yes	Yes	Yes	Weak, irregular heart sounds ascites, upper abdominal pain	No	No	1:320	Died 64th day—Postmortem, fibrinous pericarditis with effusion (1,650 cc.)
3. Stofer 1938	29 Female	Yes	Yes	Yes	Yes	Chest pain, enlarged heart, distant heart sounds, tachycardia, dyspnea, x-ray	No	Yes	1:1,280	Complete recovery
4. Foshay 1940	42 Male	None	Yes	(?) Yes	Yes	Precordial pain, dyspnea thready pulse, weak heart sounds, cyanosis	No	Yes	1:5,120	Complete recovery
5. Stump and Quinn 1940	47 Female	Yes	Yes	Yes	Yes	None	No	No	0	Died 19th day, small area of fibrinous pericarditis, aortic stenosis. Myocardial Aschoff bodies, bilateral pneumonia with pleural effusion, focal necrosis of lungs, liver and spleen
6. Kavanaugh 1941	37 Male	None	(?)	Yes	Yes	Dyspnea, cardiac enlargement, edema of legs	No	Yes	1:80	(?) Recovered
*7. Jager and Ransmeier 1943	27 Male	None	Yes	Yes	Yes	Tachycardia	No	No	1:1,280	Died 13th day. Lobar pneumonia with cavitation of right middle lobe, left lobar pneumonia fibrinous pleural pericarditis, pulmonary edema
*8. Jager and Ransmeier 1943	69 Male	Yes	Yes	Yes	Yes	None	No	No	1:20	(?) Recovered, no follow-up
*9. Jager and Ransmeier 1943	Male	Yes	Yes	Yes	Yes	Distended cervical veins, paradoxical pulse, orthopnea, dyspnea, cyanosis, ascites	No	No	0	Died 9th day. No postmortem examination
10. Jager and Ransmeier 1943	22 Male	None	Yes	Yes	Yes	Dyspnea, cardiac enlargement, distant heart sounds paradoxical pulse, tachycardia, ascites, edema, elevated venous pressure	Yes	Yes	1:1,280	Developed constrictive pericarditis with persistent cardiac embarrassment

Case Number and Author	Age Sex	External Lesion	Pneumonia	Pleural Effusion	Pericardial Rub	Other Signs of Pericarditis	Pericardial Tap	EKG Evidence of Pericarditis	Agglut.	Subsequent Course
11. Aagaard 1944	23 Male	None	Yes	Yes	Yes	Tachycardia	No	(?)	1:320	Recovered
12. Aagaard 1944	37 Male	None	Yes	Yes	Yes	Edema, orthopnea, dyspnea	No	Yes	1:2,560	Recovered
13. Morgan 1947	28 Male	Yes	Yes	Yes	Yes	Dyspnea	No	No	1:40,960	Complete recovery after treatment with streptomycin
14-24. (11 cases) Drobinskij 1947 (Describes 1 case)	23 Male	Yes	Yes	Yes	Yes	Severe substernal pain, tachycardia, low pulse pressure, dyspnea, x-ray, pericardial effusion, cyanosis, paradoxical pulse	No	(?)	1:200	Died 6th day. Postmortem-pneumonia with abscess formation, sero-fibrinous pericarditis. Bilateral pleural effusion. Myocarditis, necrosis of liver, spleen, kidneys. Abscess of 3rd finger, right hand
25. Meredith 1948	30 Male	None	Yes	Yes	Yes	Weak heart sounds, dyspnea, edema, distended cervical veins, enlarged liver	Dry tap	Yes	1:2,560	Complete recovery
26. Meredith 1948	13 Male	None	Yes	Yes	Yes	Distant heart sounds, dyspnea, orthopnea, edema, distended neck veins, tachycardia, elevated venous pressure	Yes	Yes	1:1,280	Complete recovery after treatment with streptomycin and pericardiectomy
27. Ljung 1955	51 Male	None	None	None	Yes	Chest pain, substernal	No	Yes	1:960	Recovery after treatment with streptomycin and chlortetracycline
28. Marshall, et al. 1957	42 Male	No	Yes	Yes	Yes	Diminished apical impulse	No	Yes	1:1,280	Relapse after 7 days of streptomycin. Successfully retreated with streptomycin for 10 days
29. Author 1957	59 Male	No	Yes	Yes	Yes	Substernal chest pain. Tachycardia, dyspnea, paradoxical pulse, cardiac enlargement	No	Yes	1:20,480	Recovery after treatment with streptomycin
30. Author 1957	37 Male	Yes	None	None	Yes	Severe substernal chest pain, dyspnea, cyanosis	No	Yes	1:320	Complete recovery after treatment with chloramphenicol and streptomycin

*7. Reported by Jager and Ransmeier, case from Cincinnati General Hospital.
*8. Reported by Jager and Ransmeier, case of Dr. Beers, Gallipolis, Ohio.
*9. Reported by Jager and Ransmeier, case of Dr. Swenson, Grand Rapids, Michigan.

giving 0.5 Gm. every 6 hours. It was noted that the friction rub lasted nine days. The patient became afebrile and asymptomatic on the 12th hospital day. On the 15th hospital day, a temperature of 104.2°F. was considered to be a reaction to streptomycin and the drug was discontinued. The temperature returned to normal and he was discharged on August 26, in good condition.

Review of the Literature

A summary of the pulmonary and cardiovascular manifestations of pericarditis caused by tularemia is presented in Table I. In 1929, Simpson[3] reported the first case of pericarditis due to tularemia. In 1943 Jager and Ransmier[4] in an excellent review of this subject, reported the cardiovascular manifestations in ten cases of tularemic pericarditis. In 1947, in the Russian literature, Drobinskij[1] describes one case of pericarditis due to tularemia and refers to 10 others he had seen without further description of these cases. In 1950, Meredith[12] describes two cases of tularemic pericarditis, one of which developed chronic constrictive pericarditis. In 1955, Ljung[2] describes one case of ulcero-glandular tularemia in which the only complication was pericarditis. In 1957, Marshall et al[13] reports a well-documented case of tularemic pericarditis associated with pleuro-pulmonary lesions.

Discussion

It has been postulated that tularemic pericarditis develops by direct extension from adjacent pleural involvement or from areas of pneumonitis. While this is a likely explanation, the finding of cases without apparent pleurisy or pneumonitis suggests either direct infection of the pericardium from the original inoculation site, or the presence of inconspicuous intermediary pulmonary lesions. Whatever the pathogenesis, a diagnostic problem is presented by cases of pericarditis clinically unassociated with pulmonary disease, and on some occasions without a history of exposure to rabbits. The second case in this report is an example of the latter problem and a similar case is described in the report of Marshall and Zimmerman.[13] Although pericarditis is a rare complication of tularemia the possibility of prompt, life-saving treatment emphasizes the importance of diagnosing these otherwise serious illnesses.

As emphasized by Morgan[11] in 1947, the treatment of choice is streptomycin, but Ljung's case in 1955, apparently did not respond to this drug alone, although it did respond to chlortetracycline. Meredith[12] felt that streptomycin was beneficial even when started as late as the fourth month of the disease. It has often been our practice to employ both streptomycin and a tetracycline derivative, or chloramphenicol, in patients with tularemia when they are severely ill, although proof that this is superior to one drug is not yet available.

SUMMARY

Two cases of pericarditis due to tularemia are reported. A total of 28 other cases of tularemic pericarditis were found in the world literature.

Tularemic pericarditis may occur as the only apparent complication of tularemia, a fact of importance in diagnosing certain cases of this serious illness. If treated with streptomycin, tetracycline derivates or chloramphenicol, the prognosis is similar to benign sero-fibrinous pericarditis.

RESUMEN

Se relatan dos casos de pericarditis debidos a tularemia.

Se han encontrado en la literatura mundial un total de 28 casos más. La pericarditis tularémica puede acontecer como la única complicación aparente de esa enfermedad lo que es un hecho importante para el diagnóstico de ciertos casos de esta grave enfermedad. Si se trata con estreptomicina, derivados de tetraciclina o cloramfenicol, el pronóstico es como el de la pericarditis serofibrinosa benigna.

RESUME

L'auteur rapporte deux cas de péricardite due à la tularémie. Il a trouvé un total de 28 autres observations de péricardite tularémique dans la littérature mondiale.

La péricardite tularémique peut réaliser l'unique complication apparente de la tularémie, fait d'importance dans le diagnostic de certains cas de cette affection grave. Si elle est traitée par la streptomycine, les dérivés de la tétracycline ou du chloramphénicol, le pronostic est analogue à celui de la péricardite séro-fibrineuse bénigne.

ZUSAMMENFASSUNG

Bericht über 2 Fälle von Pericarditis infolge Tularaemie. In der Weltliteratur fanden sich im ganezne 28 weitere Fälle von tularaemischer Pericarditis.

Die tularaemische Pericarditis kann als die einzige augenfällige Komplikation der Tularaemie auftreten, eine Tatsache, die von Wichtigkeit ist bei der Diagnose bestimmter Fälle dieser äusserst ernsten Erkrankung. Bei Behandlung mit Streptomycin. Tetracylin-Derivaten oder Chloramphenicol ist die Prognose gleich derjenigen gutartiger sero-fibrinöser Paricarditisen.

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CURRENT THERAPY

Renal Artery Hypertension: A Correctable Clinical Condition

Any intrinsic or extrinsic lesion that causes sufficient narrowing of a renal artery will give rise to sustained, systemic, diastolic arterial hypertension, which may be relieved by restoration of unobstructed renal blood flow or by removal of all renal tissue distal to the stenosis. This hypertension is different from that associated with atrophy of renal parenchyma secondary to primary renal disease. In renal artery hypertension the renal parenchyma is normal, and remains so unless total vascular insufficiency develops. Although suspected for years, this clinical counterpart of the experimental Goldblatt kidney has been clearly delineated only recently. In the presence of severe renal arterial stenosis parenchymal ischemia is prevented by elevation of the systemic blood pressure. This assures an adequate blood supply and preserves the renal parenchyma unless the vascular occlusion becomes virtually complete.

Atherosclerosis is the commonest cause of such stenosis.[1] It may cause primary stenosis or occlusion of a renal artery. Atherosclerosis of the abdominal aorta may secondarily involve the renal artery by occlusive plaques in the orifice, by ascending thrombotic occlusion of the aorta, by an aneurysm with secondary narrowing of the renal artery, or finally, by loosened plaques or thrombi plugging a renal artery as a result of operative procedures on the aorta.

Congenital lesions are second in importance. Aneurysms may or may not be associated with stenosis of a renal artery. Congenital narrowing of the abdominal aorta may be associated with concomitant narrowing of a renal artery. Other cases have been reported as a result of trauma, embolic infarction, external compression of a renal artery, and miscellaneous primary lesions of the artery itself.

The most perplexing problem is the difficulty of ready recognition of the condition. Fixed, usually progressive, diastolic hypertension occurs with some or all of the usual manifestations of severe essential hypertension. Symptoms may be severe and associated with an overwhelming inner tension and restlessness. Urinary symptoms are exceptional. The size of the heart and electrocardiographic observations are consistent with the duration and severity of the hypertension. Results of urinalysis are normal unless renal infarction has occurred recently. Results of common renal function studies are normal and intravenous and retrograde pyelography reveal normal kidneys, or slight decrease in the size of the affected kidney.

Special diagnostic technics are essential. Arteriography is the most useful of these. The vessels must be clearly defined and severe narrowing accurately demonstrated. With proper precautions aortography can be

safely performed and it is indicated if hypertension of the renal artery is suspected. Contraindications to its use are severe bilateral renal parenchymal disease and sensitivity to organic iodine compounds. In the adult translumbar aortography with use of heavy sedation and local infiltration with procaine is preferred. In children and nervous adults intravenous administration of pentothal is used. In the small child retrograde femoro-aortic catheterization is performed. We prefer small doses of a 50 per cent solution of sodium diatrozoate (Hypaque®), repeated, if necessary, to outline all renal arteries sharply. An injection of 10 ml. of a 0.5 per cent solution of procaine through the arterial needle just before the dye is injected minimizes arterial spasm and reduces pain. Careful study will demonstrate transient subclinical changes in renal plasma flow but we have not experienced any serious or persistent complications. Postoperative arteriography is indicated as a check on the reconstructive surgical repair.

Characteristic observations have been obtained on careful differential renal clearance studies of the glomerular filtration rate (inulin clearance) and the renal plasma flow (PAH clearance). From these is calculated the tubular rejectate fraction of sodium. On the affected side the urinary output, renal plasma flow, glomerular filtration rate, tubular rejectate fraction for sodium and chloride, and the Tm_{PAH} are decreased. The inulin U/P ratio is increased. On the other side the tubular rejectate fraction for sodium and water is increased and the inulin U/P ratio is decreased.

Both of these diagnostic methods are too formidable for routine tests. Scintillation scanning of both kidneys, after intravenous injection of appropriate radioactive materials, may prove to be a useful screening method. A few cases have been reported in which decrease in the renal blood flow was detected by this means.[2] However, more evidence is needed. Our differential renal clearance studies indicate that reduction in the renal plasma flow on the affected side may not be striking. If this is true, the radioactive scanning technic may not prove entirely reliable. Evidence of reduced blood flow should stimulate definitive studies, but lack of such evidence does not necessarily rule out significant arterial stenosis. A simple reliable screening technic is urgently needed. The diagnosis is definitely established only at operation with demonstration of a significant drop in blood pressure across the area of stenosis.

Renal arterial stenosis should be suspected if hypertension appears, in the absence of primary renal disease, before the patient is 30 years old or after the age of 50 years; if a murmur is transmitted to the femoral region; if there is an unexplained difference in size or function of one kidney; if the hypertension suddenly worsens; or if the natural history of the hypertension is atypical. In such cases definitive diagnostic procedures are indicated.

Surgical correction of renal arterial stenosis is indicated, in the absence of severe medical contraindications, whenever serious hypertension is apparently due to stenosis of a renal artery. In the elderly the danger of development of coronary or cerebrovascular insufficiency with reversion to

normotension must be considered. This accounted for the only death among our eight patients. Vascular reconstruction is preferable to renal ablation and is indicated whenever it is technically possible and clinically feasible. If the contralateral kidney has good function, a severely damaged kidney should be removed, even an undamaged kidney should be removed whenever vascular reconstruction is technically impossible or the general condition of the patient contraindicates the somewhat longer reconstructive operation.

A long midline abdominal incision is distinctly desirable for several reasons. It provides adequate exposure of both renal arteries, and permits exploration and biopsy of both kidneys as well as exploration of both adrenal glands and the abdominal aorta. The following operative examinations are important:

1. Determination of the size and configuration of both kidneys.
2. Biopsy of both kidneys. Significant atrophy of the ipsilateral kidney is an indication for nephrectomy. In older patients demonstration of nephrosclerosis predicts some postoperative systolic hypertension.
3. Confirmation of absence of a renal tumor.
4. Observation and palpation of the abdominal aorta and both renal arteries.
5. Measurement of the intra-arterial pressure proximal and distal to the point of stenosis. Unless a significant fall in blood pressure is present distally, the diagnosis is not established.
6. A second measurement of the arterial pressure proximal and distal to the point of vascular reconstruction is essential to check the adequacy of the new lumen.

The type of vascular reconstructive procedure employed depends on the lesion encountered. With extrinsic compression obviously the obstructing lesion should be removed. In atherosclerosis, endarterectomy has proved satisfactory. We prefer to do this through a transverse incision in the renal artery just distal to the sclerotic area. With exclusion of that portion of the abdominal aorta, proximal endarterectomy may be performed carefully. To prevent anastomotic stenosis the artery is reunited with interrupted simple end-on sutures of fine arterial silk. In some cases of congenital stenosis the involved segment may be excised with end-to-end reconstruction of the renal artery. More commonly the stenosis is juxtaaortic and primary reconstruction requires the difficult and sometimes unreliable reimplantation of the renal artery into the aorta. On the left side the splenic artery lies within a few millimeters of the renal artery and the blood flow may be satisfactorily restored by division of the splenic artery and swinging it down for anastomosis to the renal artery. We prefer end-to-side anastomosis because the natural channel is preserved, and general experience with peripheral vessels indicates that such anastomoses are less apt to stenose than end-to-end ones. Under certain circumstances foreign grafts must be used but we avoid them, if possible, particularly in the younger age group, because of the common late development of stenosis in such grafts in the lower extremities. If the blood supply and function of the

opposite kidney are good, nephrectomy is a reliable though destructive method of correcting renal artery hypertension.

The reports of other,[1,3] as well as our own experience with 3 patients treated by nephrectomy and 5 patients treated by vascular reconstruction,[4] indicate that prompt lasting relief of the hypertension with its attendant symptoms may be achieved, either by appropriate renal ablation or adequate vascular reconstruction. It must be re-emphasized that operative demonstration of a significant fall in the intra-arterial pressure across the area of stenosis is essential to establish the diagnosis, and absence of such a pressure differential after vascular reconstruction is essential to attest its adequacy. Under these circumstnces, in the absence of other causes of hypertension, such as nephrosclerosis, complete lasting relief of the hypertension may be confidently anticipated.

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ELECTROCARDIOGRAM OF THE MONTH

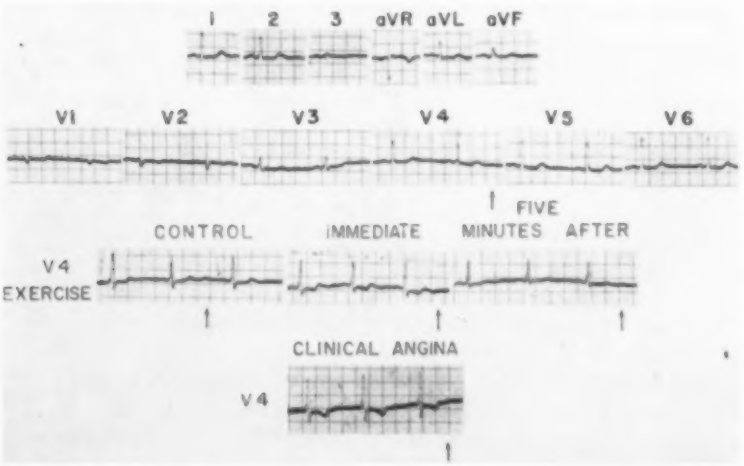
The authors would be pleased to receive comment and controversy from readers in relation to explanations offered.

The Inverted U Wave

Mrs. M. G. is a 56 year old office assistant who was first seen on August 8, 1954. She gave a proper description of the angina syndrome of one month's duration. She had had a right-sided sympathectomy for peripheral arteriosclerosis obliterans in 1950. Her blood pressure was 124/74. Her mother had died at the age of 42 years of hypertension and a stroke. Her 12 lead ECG (see illustration) showed depressed ST segment in Lead aVL and small upright T waves in V2 and V3. On August 25, 1954 the T waves were inverted in Leads VI-V4 (without Q waves in those leads) and the ST segment of Lead V4 was depressed (see "Clinical Angina" in illustration). During this time, her anginal symptoms were relieved by nitroglycerine. On September 25, 1954 the precordial T waves were still inverted but much less so and the ST segment in Lead V4 had become normal. On October 25, 1954 her tracing was similar to the 12 lead ECG shown in the illustration and has remained so up to the present time. Her anginal symptoms have decreased significantly since 1956.

On or about November 1, 1954, a physician in the patient's family performed a 2-step exercise test (without the author's knowledge); this is labelled "Exercise" in the illustration. Note that the control U wave in Lead V4 (as well as in the 12 lead ECG) is upright. Immediately after exercise the ST-T complex became of the ischemic type and was followed by an inverted U wave. Five minutes after exercise the U wave was upright and normal. During an attack of clinical angina on August 25, 1954 (see illustration), the U wave was diphasic with an inverted initial phase.

The U wave is considered abnormal when inverted, diphasic, or upright but taller than 2 mm. It is known that inverted U waves occur most commonly in hypertension, but also in coronary sclerosis and left ventricular hypertrophy; in acute myocardial infarction they undergo an evolution, as the author has shown.



independent of the T wave evolution. Inverted U waves may also occur as the sole abnormality in an otherwise normal ECG, but this is noted more often in hypertensive cardiac disease and in coronary sclerosis.

In this patient, the U wave became inverted or abnormal following either clinical or induced anginal manifestations, and reverted to upright or normal following subsidence of the myocardial ischemia. The inverted U wave is a valuable, although infrequent, corroborative sign of myocardial ischemia.

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Sicklemia Complicating Chest Surgery

A Case Report

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The problems confronting the patient with sickle cell anemia have become well known. Only recently, however, has it been accepted that sickle cell trait *per se* (sicklemia) is capable of producing clinical symptoms and pathological findings.[7, 10, 15] The development of conditions favorable to sickling, such as factors producing anoxemia and stasis, may precipitate symptoms in the otherwise asymptomatic patient. Sicklemia may pass unrecognized in the routine preoperative evaluation of the patient, because only one in 15 cases show any anemia.[12]

The purpose of this paper is to point up this problem as it applies to the preoperative evaluation for chest surgery. The following is a report of sickle cell crisis during surgery in a patient with sicklemia in whom additional features of an unusual nature were encountered.

A 17 year old Negro was admitted with a chief complaint of hemoptysis. His subjective symptoms had started six months prior to admission with the onset of recurrent cough productive of mucoid sputum. One week prior to admission he experienced hemoptysis of bright red blood, resulting in hospitalization.

Systemic review was essentially negative except for intermittent left chest wheeze for the past six to seven years. There was no history of dyspnea or chest pain.

Past history revealed pertussis at the age of two years; frequent upper respiratory infections; and pneumonia at the ages of six, nine, and 11 years. Each occurrence of pneumonia required hospitalization, the longest of which was two and a half months at the age of nine years. No previous history of sickle cell crisis was elicited.

Physical examination revealed an asthenic 17 year old Negro who complained of nonproductive cough. His blood pressure both arms was 108/72, pulse 84 and regular, respirations 16 and weight 148 pounds. Positive physical findings consisted of prolonged expiratory phase of the left hemithorax with resulting left chest lag; hyperresonant left hemithorax to percussion; and crepitant inspiratory post-tussic rales over the left lower lobe area.

Laboratory data consisted of 5900 white blood cells with 51 per cent polys, 47 per cent lymphocytes, one monocyte, and one eosinophil and 5.3 million red blood cells. Hemoglobin was 15.0 grams per cent. Erythrocyte sedimentation rate was 2 mm./hr. Hematocrit was 50 volumes per cent. Sickle cell preparation revealed 30 per cent sickling in six hours. Hemoglobin electrophoresis showed 80 per cent A hemoglobin and 20 per cent S hemoglobin, indicating sicklemia. Urinalysis was negative. Blood urea nitrogen was 14.9 mgms. per cent. Fasting blood sugar was 81 mgms. per cent. Serologic test for syphilis was negative. Purified protein derivative, histoplasmin, and coccidiodin first and second strength skin tests were negative.

Electrocardiogram was within normal limits with an intermediate axis. Postero-anterior inspiratory roentgenogram of the chest (Figure 1) demonstrated marked increase in radiability on the left and unduly prominent lung vascular markings in the right hilar and paracardiac regions.

Postero-anterior expiratory roentgenogram of the chest (Figure 2) revealed the maintenance of the inspiratory volume of the left lung with resulting marked shift of the mediastinum to the right. Fluoroscopy showed the expiratory lag of the left diaphragm to be in phase with the mediastinal shift.

Bronchogram (Figure 3) revealed a normal right pulmonary tree. The left lower

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The Opinions expressed are those of the authors and do not necessarily represent the views of the Bureau of Medicine and Surgery, Navy Department.

lobe showed cylindrical and saccular bronchiectatic changes probably resulting from previous infections superimposed upon the mal-developed left lung.

Angiocardiography (Figure 4) showed the hypoplastic left pulmonary artery with small but normally distributed segmental divisions. The right pulmonary vascular

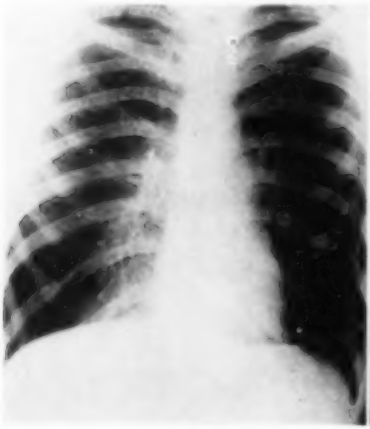

FIGURE 1

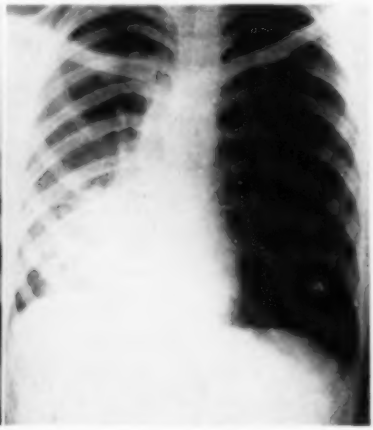

FIGURE 2

Figure 1: Postero-anterior inspiratory roentgenogram of the chest demonstrates the increased radiability and scarcity of lung vascular markings in the left hemithorax.—*Figure 2:* Postero-anterior expiratory roentgenograms of the chest. Note the marked shift of the mediastinum to the right.

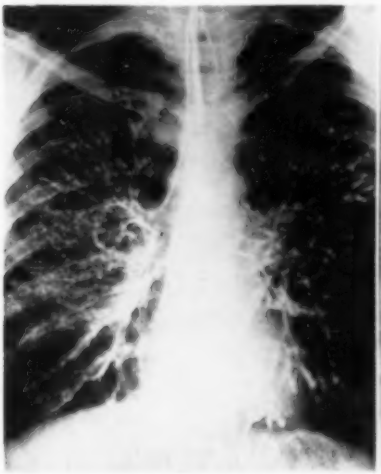

FIGURE 3

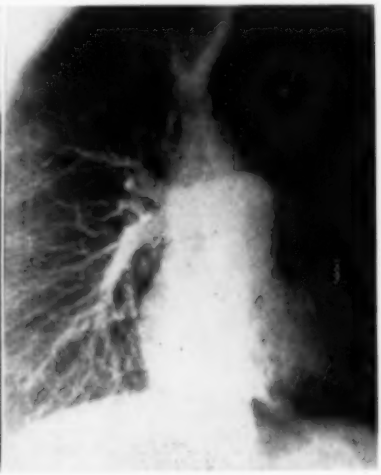

FIGURE 4

Figure 3: Bronchogram in the postero-anterior projection illustrates the normal right bronchial tree with bronchiectatic changes in the widely spaced left lower lobe bronchi.—*Figure 4:* Angiocardiogram demonstrates the marked hypoplasia of the left pulmonary artery with the usual segmental divisions. The vessels in the right lung are widened suggesting increased blood flow.

tree was enlarged but normally segmented. No definite aberrant, systemic, blood supply to the left lung was demonstrated.

Bronchoscopy revealed diffuse inflammation throughout the left main stem bronchus. Pulmonary function tests showed uneven alveolar mixing and an air velocity index of .84; otherwise all other values were within normal limits.

In order to further differentiate pulmonary function, right and left lung bronchospirometry was done (Table I) revealing a gross ventilatory defect of the left lung. Arterial blood study showed no evidence of oxygen desaturation or carbon dioxide accumulation with exercise.

Thoracotomy was elected to determine if surgical benefit could be obtained. On entry into the left chest a pale left lung with little pigment was found. The left lower lobe was fixed by what appeared to be inflammatory adhesions to the diaphragm and posterolateral chest wall. Pulmonary function studies at the time of operation revealed poor aeration of the left lung which was able to empty only 150 cc. of air by its own compliance. The pulmonary artery was hypoplastic and less than 50 per cent of its normal caliber. An anomalous pulmonary artery, about 6 mm. in diameter, was found in the inferior pulmonary ligament coming from below the diaphragm. Emphysema was diffuse and not localized. He tolerated anesthesia poorly with early appearance of tachypnea, pink facies and precipitious rise of blood pressure to 188/120. Lung biopsy was obtained and thoracotomy judiciously terminated.

Post-operative blood studies showed sickling and drop of hemoglobin to 9.5 grams. By exposing an unsealed, wet film of the blood to an atmosphere of 18 per cent oxygen and 10 per cent carbon dioxide 100 per cent sickling in two minutes was produced post-operatively.

Discussion

In vitro sickling has been shown to depend on the dissociation of oxyhemoglobin in the red blood cell. When hemoglobin is in the reduced state, the cells sickle. When the cells combine with oxygen, they resume their normal discoid shape. An oxygen tension of 45 mm. Hg. has been reported to produce sickling in cells from a patient with sickle cell anemia, while it was necessary to reduce the tension to 18 mm. Hg. in the case of cells from a person with sicklemia. A lowering of blood Ph likewise has been found to favor sickling. The danger of airplane flight and lowered oxygen tension to persons with sicklemia has previously been reported.[15] During surgery the flushed, pink facies, and precipitous rise in blood pressure are best explained by hypercapnia[3] caused by obstruction to respiration or narcotic depression of it. The patient most likely sickled at this point further hindering pulmonary blood flow and producing a vicious cycle of hypoxemia and sickle cell crisis. This sequence of rapidly occurring events endangered the patient and the operative procedure had to be terminated.

The presence of sicklemia adds an additional risk to chest surgery. In the susceptible patient sickle cell tests and hemoglobin electrophoresis should be done in order to better evaluate operative risk.

TABLE I

Bronchospirometry Per Cent	Right Lung Per Cent	Left Lung Per Cent
Min. Ventilation (52-58)	83	17
Min. Oxygen Uptake (52-58)	83	14
Ventilatory Equivalent (1.1 to 1.3)	1.90	2.39
Vital Capacity (52-58)	68	32
Residual Volume (52-58)	68	32

The association of anomalous pulmonary arteries with bronchiectasis, pulmonary cysts and intra-lobar sequestration has been well documented[4, 14] and embryology has been described.[1, 11] The role that anomalous pulmonary arteries may play as a cause of dyspnea, recurrent hemorrhage or recurrent infection has been suggested by Mair.[13] This condition is not sufficiently appreciated by the internist, and the diagnosis is rarely made preoperatively. Accidental transection of an anomalous pulmonary artery during surgery has resulted in three deaths from hemorrhage.[2, 6, 9] Angiocardiography[5, 8] has, on occasion, demonstrated anomalous pulmonary arteries and has become of practical importance in preoperative evaluation.

The role that the anomalous pulmonary artery may have played as a cause of hemorrhage in this patient is open to speculation.

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Editorial

Look Again and Again and Again

With approximately one-third of the 173 million people of the United States harboring tubercle bacilli, complacency has developed to such a degree that many sanitoriums have been closed and individuals and organizations are devoting funds, time, and effort to other diseases; all of which is causing complacency to mount. With only 0.156 per cent of the 95 million cattle in this country infected with tubercle bacilli veterinarians and their allies are warning against complacency and are tightening their belts and employing their most powerful weapons in an all-out drive to eradicate the disease from cattle.

In this issue of *Diseases of the Chest* Dr. A. F. Ranney, Chief, Tuberculosis Eradication Section, Animal Disease Eradication Division, United States Department of Agriculture, warns against complacency. He says, "The fight against bovine tuberculosis is not over until the last source of infection is wiped out . . ." He points out that with the fine accomplishments to date, "The plain truth is that bovine tuberculosis is still with us and we haven't won the fight or done our job until we've mopped up the last traces of it."

The accomplishments of veterinarians and their allies in the United States has been designated "Man's Greatest Victory Over Tuberculosis" for the reason that no report has been found of such a large area with so many animals or people involved with such fine achievements. This accomplishment by veterinarians is the result of thoroughly familiarizing themselves with the tubercle bacillus and its behavior in the tissues of animals. No sooner was tuberculin presented to the world by Koch than veterinarians put it to the most rigid test by making post mortem examination of the carcasses of reactors and non-reactors to tuberculin. They found the test was a very accurate procedure for determining the presence of tuberculosis in the animals body. Since the first veterinary school was established in Lyons, France in 1762, a tremendous amount of work was done on tuberculosis in cattle, but nothing of importance was accomplished until the tuberculin test became available which with great accuracy detected the presence of tubercle bacilli in the apparently healthy as well as ill animals. Thus they were able to remove from herds animals harboring tubercle bacilli long before these organisms had time to produce serious and contagious disease or transmit organisms to others. Up to that time the attack had been made on the damage done by tubercle bacilli. Thereafter the attack was made directly upon the tubercle bacillus with the aim to reducing its numbers ultimately to the vanishing point. This goal appeared within the realm of physical possibility, therefore a national program was instituted in 1917. This included going from farm home to farm home, township by township, county by county, and state by state, periodically. The single animal farm was included the same as that with many animals so that no reactor would be missed. It was not just one

nation-wide testing but repetition until the problem could be solved. There were persons who thought the solution was only a few years off, however those who had a better understanding of tuberculosis girded for a long battle. For example, in 1917 Dr. J. A. Kiernan, first Chief of the Tuberculosis Eradication Division of the Bureau of Animal Industry, said: "There absolutely are no grounds upon which a reasonable estimate can be made of the number of years it will take to eradicate this disease. All one can do is to make a guess as to the time, and, it is my belief that if this nation succeeds in eradicating tuberculosis in fifty years, it will be one of the greatest heritages our successors will have handed down to them."

From 1917 to 1957, 378,803,473 tuberculin tests were administered to the cattle of this country by the Animal Disease Eradication Division of the United States Department of Agriculture and State Livestock Sanitary Boards. This resulted in finding 4,062,634 animals harboring tubercle bacilli. This is the largest experience in tuberculin testing with post mortem examination of reactors ever reported. It justifies the statement that veterinarians know more about every aspect of tuberculin testing than physicians in human medicine or any other group of workers.

Tuberculosis in cattle is no different than that in people, in the allergy it produces, the pathology it manifests, and its epizootiology. Therefore, the tuberculin test finds people harboring tubercle bacilli just as it does cattle. The tuberculin test is an accurate procedure for determining the presence of tuberculosis in people the same as it is in cattle. These facts were established by Ghon who between 1908 and 1912 did meticulous post mortem examinations on 184 children who had died from non-tuberculous conditions and presented no evidence of tuberculosis except tuberculin reaction during life. The object of his study was to determine whether the tuberculin test is specific in people. Among the 184 bodies he found lesions in 183; the remaining body was not completely examined. In his report he said, "From the standpoint of the pathological anatomist my researches, which in regard to the question are not limited to the cases described here, justify me in concurring entirely with those who advocate the specific value of the tuberculin reaction."

In making post mortem examinations of so many tuberculin-reactor cattle, occasionally lesions were not found. At first these were designated "no-lesion reactors," but this term was later changed to "no-visible-lesion reactors" because it was thought that lesions were present, but because of their size or unusual location they were missed on routine necropsy. More recently preference has been given to the term "no gross lesion found." In 1923 Calmette said, "Tuberculin has frequently been accused of having given a false indication because no tuberculous lesions could be found." "It was proved however long ago that in these circumstances the organs had not been searched with sufficient care." "Whenever a tuberculin reaction is positive, there exists somewhere a follicular lesion or at least a gland containing tubercle bacilli whose presence can be disclosed by experimental inoculation of guinea pigs." In 1942 Schroeder cited a number

of instances in which the tuberculin reaction among cattle was not explained until long and tedious post mortem examination, not possible in slaughter houses, revealed tuberculous lesions in unusual and unexpected locations.

Further evidence that lesions are present but are missed in the routine necropsy of so-called "no-gross-lesion-found" reactors was adduced and reported by Nassal in 1956. Among a thousand cattle carcasses by meticulous post mortem examination, he found unmistakable evidence of tuberculosis in 50.2 per cent of those designated no-visible-lesion reactors on routine postmortem examination. The question is now being asked if even more meticulous examination would not have uncovered lesions in the remaining 49.8 per cent of these animals.

Members of the veterinary profession and their allies have led the way and have established facts which must be employed in tuberculosis among humans if this disease is to be eradicated. When this statement is made, the old, old retort is still heard, "You can't slaughter people." To my knowledge, no such suggestion has ever been made. However, procedures have been devised for managing persons who react to tuberculin which, in the long run, are just as effective in reducing the populations of tubercle bacilli as the veterinarians' method. The fact remains that the goal will never be attained among human beings unless those harboring tubercle bacilli are identified. This can be done only by the tuberculin test.

Physicians in human medicine and their allies must catch the same vision of tuberculosis eradication as veterinarians have long had and participate in a program that consists of contacting every home, locating all persons harboring tubercle bacilli, and acting accordingly. When veterinarians proposed such a program for the cattle of the entire nation, it was often referred to as an idle dream, but it succeeded. When this procedure is proposed for people, it is referred to as an idle dream-Utopian. Despite the complacency that had to be overcome and the word "perfectionists" often used in a derogatory way, the method has been thoroughly demonstrated and its efficacy so proved that it can be highly recommended everywhere.

Only a few decades ago physicians in human medicine were critical of veterinarians because animals were infecting people with tubercle bacilli. Today veterinarians can be critical of physicians for allowing people to infect cattle with tubercle bacilli. This is occurring so often that periodic tuberculin testing of cattle is now recognized as a case finding method among people.

Inasmuch as the three well-known types of pathogenic tubercle bacilli, human, bovine and avian, produce progressive disease in more than one species and they all cause clinical disease in people, it is obvious that the attack on tuberculosis must be on all three types of tubercle bacilli. This necessitates the closest cooperation between veterinarians and physicians. As long as animals can transmit tuberculosis to people and vice versa, there will be no hope of eradicating tuberculosis while any one of the three types of pathogenic organisms is in existence.

Veterinarians have so much to contribute from their extensive experience and accomplishments that their profession should be represented on every board of health and every tuberculosis organization. Moreover, their writings should not be limited to veterinary journals, but should appear frequently in medical, nursing, and public health publications.

The article in this issue by Dr. Ranney can be read with great profit by physicians around the world. It not only calls attention to what has been necessary by way of diagnosis, epizootiology, et cetera, in bringing about the finest accomplishment of all time, but emphasizes especially the imminent danger of complacency which could result in loss of all that has been gained.

The danger of such complacency as now exists with reference to tuberculosis in people is evident. There is no fact to justify the slightest degree of complacency, since in this country there is 211.5 times more tuberculosis percentagewise in people than in cattle. Among the 2,700,000,000 citizens of the world, tuberculosis causes more serious incapacity and death than any other communicable disease, and in some places, more than all others combined. Even with such facts confronting us, complacency has reached a dangerous level and relaxation of effort is in evidence everywhere. Tubercle bacilli are so abundantly present among our citizens that if the reins are not promptly tightened and an all-out offensive attack made on the organism, the disease can again become as destructive as it was at the opening of the Twentieth Century.

J. ARTHUR MYERS, M.D., F.C.C.P.
Minneapolis, Minnesota

Professor Hugo Wilhelm Knipping.

PRESENTATION OF THE INTERNATIONAL COLLEGE MEDAL

It is with deep humility that I take on this unique and auspicious assignment before this magnificent gathering and in the presence of a veritable galaxy of brilliant minds.

It has been one of the traditional functions of the College to select men from time to time for a certificate of merit and for the College medal for distinguished contributions to science. Over a year ago, the search began for a man who fulfilled stipulations set up for adding a new name to the Hall of Fame of the American College of Chest Physicians. It has been our good fortune to find the man who has become widely known for his ability and talent for solving scientific problems with as much ease, ingenuity and daring as Alexander the Great solved the Gordian knot. He is a scientist of first magnitude and a mastercraftsman of research. He is a man of intuitive intellect, disciplined imagination and profound knowledge. These are the ingredients which led him to discovering new scientific methods and instruments for exploring the workings of the human body. His precision methods in determining the functional capacity of the heart, the blood circulation and the lung contributed tremendously to progress in the pertinent specialties. Industrial medicine as well as practice of medicine at the bedside derived great benefits from his scientific innovations. Among other items, he is noted for introducing radioactive isotopes for use in the diagnosis of carcinoma of the lung, bronchial occlusion, atelectasis and coronary insufficiency.

This man is a teacher of medicine of great renown. In his famous institution students and doctors of medicine gather from all over the world. They go there not only to gain new knowledge but also to acquire inspiration and enthusiasm with which he is imbued. Moreover, they like myself, enjoy basking in the warmth of his sparkling wit and wisdom.

Last but not least, this man is—in the true, ideal sense of the word—a good physician. A good physician makes an accurate diagnosis and administers the best remedies. But this is not all. A good physician, according to classical Greek concepts, is like a god, because a good physician not only treats his patient but also understands him, counsels him and, what is most important, has compassion for him.

All this being so, Ladies and Gentlemen, it is my great pleasure and privilege to present to you the recipient of the international medal of the American College of Chest Physicians, the illustrious Professor and Chairman of the Department of Medicine at the Medical School of the University of Cologne, Germany, Dr. Hugo Wilhelm Knipping.

Andrew L. Banyai, M.D., F.C.C.P.
Chairman, Council on International Affairs

Delegates in attendance at the Fifth International Congress, Tokyo, Japan, September 7-11, 1958. This photograph was taken on the roof of the Daiichi Building, headquarters of the Congress. The Imperial Palace, its gardens and moat, can be seen in the background.

FIFTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The Fifth International Congress on Diseases of the Chest, held in Tokyo, Japan, September 7-11, was an outstanding success. The congress was sponsored by the Council on International Affairs of the American College of Chest Physicians and presented under the auspices of the Japanese Government, the Japan Medical Association, the Japan Science Council and the Japan Chapter of the College.

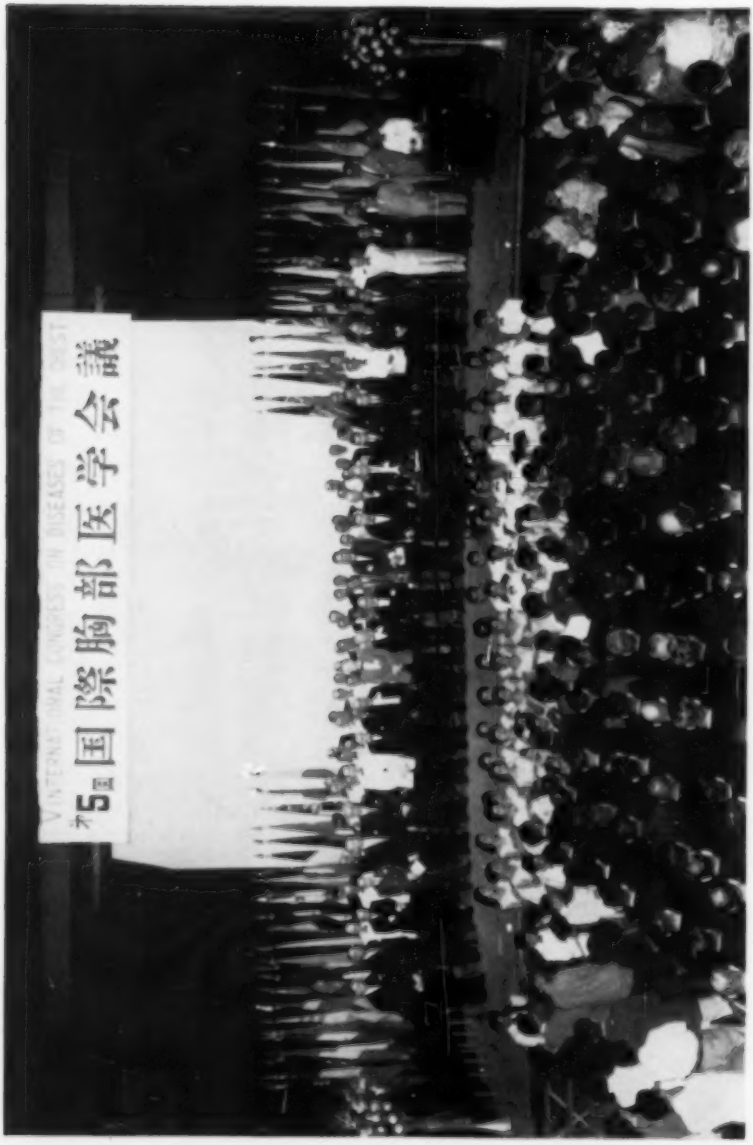
Registration for the congress reached a total of 1,268 physicians and their families representing thirty-five countries and territories throughout the world. The arrangements for both the scientific and social activities were splendid. Without minimizing the success of our previous international congresses, all of which were excellent, this one was the most efficiently organized. Much of the credit for its success goes to Dr. Jo Ono, Secretary-General of the congress and Regent of the College, Prof. Yoneji Miyagawa, Vice-President and chairman of the committee on finance, Prof. Taizo Kumagai, the President of the congress, and their staff of tireless and efficient workers. They were ably assisted by Professors Shiota, Katsunuma, Imamura, Kuno, Terada, Tsuzuki, Kawai, Kitamoto, and many other College officials and members in Japan, too numerous to name individually in this condensed report. All of them are to be commended for their efforts and generous cooperation.

Inaugural Ceremony

The congress was officially inaugurated with an impressive ceremony in the Yomiuri Hall on Sunday evening, September 7. Dr. Jo Ono, Secretary-General of the Congress, was the presiding officer. His Excellency, the Honorable Nobusuke Kishi, Prime Minister of Japan and the Honorary President of the Congress, formally opened the congress and welcomed the assembled delegates. He was presented with a special plaque in appreciation of his support to the congress and that of the Japanese Government. Greetings were also extended by the Honorable Governor Seiichiro Yasui of Tokyo, Dr. Taro Takemi, President of the Japan Medical Society, and Prof. Yoneji Miyagawa, Vice-President of the congress.

Prof. Taizo Kumagai, President of the congress, in his talk emphasized the objectives of the congress and the work to be accomplished during the days of

Prime Minister Kishi arriving at the Inaugural Ceremony, where he delivered a message to the congress delegates.



The Inaugural Ceremony, Sunday, September 7, 1958, Yomiuri Hall, Tokyo.

the scientific assembly. Dr. Seymour M. Farber, President-Elect of the College, responded in the absence of Dr. Donald R. McKay, President.

A stamp commemorating the congress was presented to Prof. Kumagai by Mr. Yutaka Terao, Minister of Posts and Telecommunications. This is the first time in the history of Japan that a commemorative stamp was issued for a medical congress. The stamp features a stethoscope, in honor of Laennec whose portrait appears on the College Seal.

More than one hundred Fellowship Certificates were presented by Dr. John F. Briggs, chairman of the Board of Regents.

The College Medal for meritorious achievement in diseases of the chest was presented to Prof. Hugo W. Knipping, Director of the University Clinic, Cologne, Germany. Dr. Andrew L. Banyai, chairman of the Council on International Affairs, made the presentation and praised Prof. Knipping for his outstanding contributions to medical science and his devotion to the alleviation of human suffering.

The closing talk was given by Mr. Murray Kornfeld, Executive Director of the College, who spoke of the progress of the College during the past twenty-five years and the value of international congresses in bringing about world peace and understanding.

Dr. Otto L. Bettag, Regent of the College and the representative of the Honorable William A. Stratton, Governor of the State of Illinois, presented a portrait of Abraham Lincoln to Prime Minister Nobusuke Kishi on behalf of Governor Stratton. Official letters from Governor Stratton and Mayor Richard J. Daley of Chicago were presented and the following message from the Honorable Dwight D. Eisenhower, President of the United States of America, Washington, D.C., was read:

Dr. Donald R. McKay, President
American College of Chest Physicians
112 East Chestnut Street
Chicago, Illinois

To all attending the Fifth International Congress on Diseases of the Chest I send greetings.

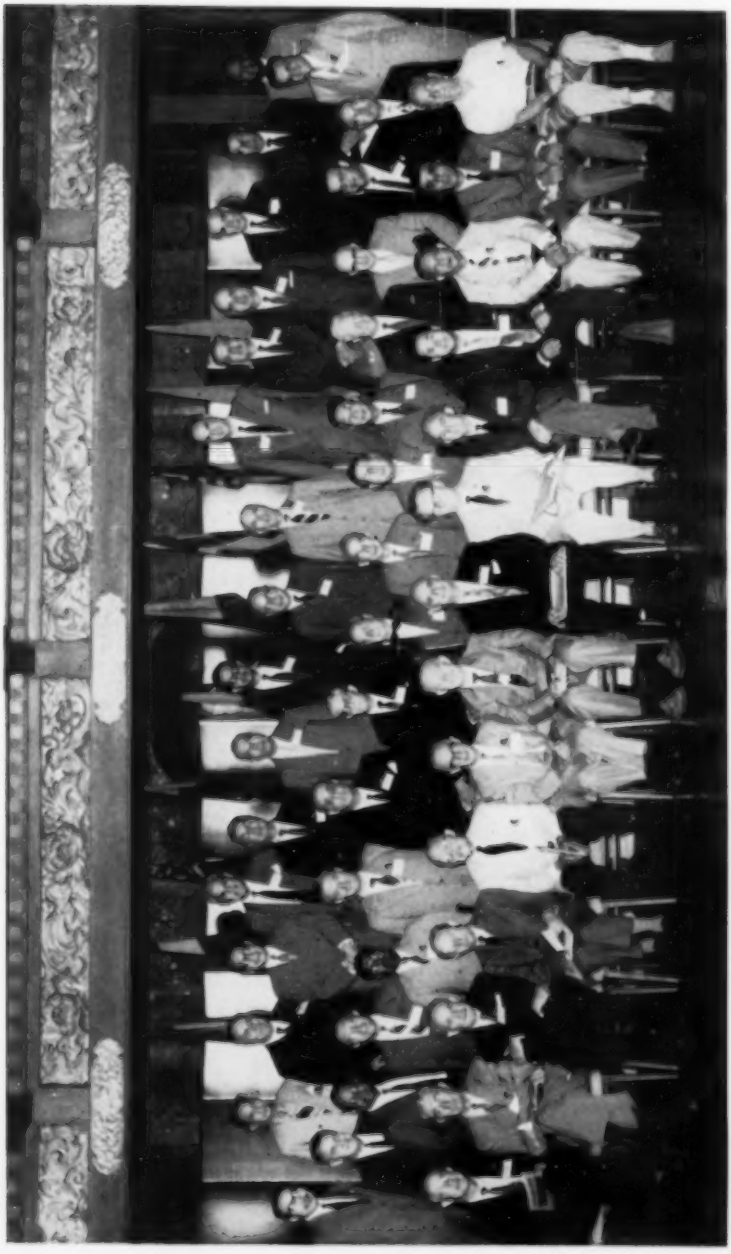
Coming together from all parts of the world and united in a common devotion to the ideals of the medical profession, your delegates can contribute much to the healing of mankind. International conferences such as yours provide an invaluable opportunity for the exchange of scientific information and are an important means of building good will and understanding among all peoples.

Best wishes for a memorable meeting.

Dwight D. Eisenhower

The Nippon Chamber Music Orchestra, under the direction of H. Iwaki, and the Tokyo Mixed Choral Group presented several numbers, including a magnificent rendition of Handel's Halleluiah.

The beautiful kimonos and obis worn by the Japanese ladies, mingled with other colorful Oriental and Western style evening dress, added a true international flavor to this Inaugural Ceremony.



The Regents and Governors of the College who attended the executive sessions of the Tokyo congress.

Executive Sessions

The opening Executive Session for Regents and Governors of the College was held at the Tokyo Kaikan, the morning of September 6. Dr. Seymour L. Farber, President-Elect of the College, presided. The following reports were presented.

Message of Welcome
Taizo Kumagai, Sendai, Japan, President of the Congress

Report of the Committee on Arrangements for the Congress
Jo Ono, Tokyo, Japan, Secretary General of the Congress

Report of the Council on Pan-American Affairs
Jose Ignacio Baldo, Caracas, Venezuela, Chairman

Report of the Council on European Affairs
Attilio Omodei Zorini, Rome, Italy, Chairman

Report of the Council on Pan-Pacific Affairs
Miguel Canizares, Quezon City, Philippines, Chairman

Report of the Council on African and Eastern Affairs
Raman Viswanathan, New Delhi, India, representing
David P. Marais, Cape Town, South Africa, Chairman

Report of the Council on International Affairs
Andrew L. Banyai, Chicago, Illinois, USA, Chairman

Report of the Editorial Board for *Diseases of the Chest*
J. Arthur Myers, Minneapolis, Minnesota, USA, Editor-in-Chief

Report of the Committee on Motion Pictures
Paul H. Holinger, Chicago, Illinois, USA, Chairman

Report of the Committee on Essay Awards
Donato G. Alarcon, Mexico City, Mexico

Report of the Committee on Resident Fellowships
Alfred A. Richman, New York City, USA, Chairman

Report of the Committee on International Insurance
Charles K. Petter, Waukegan, Illinois, USA, Chairman

Report on College Books
Coleman B. Rabin, New York City, USA

Report of the Executive Director
Murray Kornfeld, Chicago, Illinois, USA

The following resolutions were adopted:

WHEREAS, one of the main objectives of the College is to promote and improve the teaching of diseases of the chest in medical schools throughout the world, and

WHEREAS, in each country the College has members who are affiliated with medical schools and universities,

BE IT RESOLVED that the International Committee on Undergraduate Medical Education conduct a survey of the teaching of chest diseases in the medical schools throughout the world and endeavor to improve the methods of teaching and the curricula in the specialty.

WHEREAS, one of the major objectives of the American College of Chest Physicians is the promotion of postgraduate medical education, and

WHEREAS, postgraduate courses in diseases of the chest have been sponsored by the College in the United States for several years, and

WHEREAS, the College can now provide a list of approved motion pictures for use in teaching chest disease,

BE IT RESOLVED that College chapters in all countries be encouraged to present postgraduate courses in diseases of the chest, under the supervision of the Regent or Governor in each country.

WHEREAS, the Committee on Nonsurgical and Drug Therapy of the College has conducted a study of the treatment of uncomplicated pulmonary tuberculosis over a ten-year period, and

WHEREAS, this study has also been conducted in Germany and Japan under the direction of College members, the results of the Japan study having been printed in book form and distributed,

BE IT RESOLVED, that the International Committee on Nonsurgical and Drug Therapy urge that this study be undertaken in other cities throughout the world.

WHEREAS, the Section on Cardiovascular Surgery of the Committee on Cardiovascular Disease of the College has now completed studies on "The Tetralogy of Fallot," "Surgical Treatment of Coarctation of the Aorta," "Surgery of Patent Ductus Arteriosus" and "Surgical Treatment of the Tetralogy of Fallot," and

WHEREAS, the reports of these studies have been published in *DISEASES OF THE CHEST* and have proved to be of interest to physicians throughout the world,

BE IT RESOLVED, that the International Committee on Cardiovascular Surgery recommend to the Section on Cardiovascular Surgery other studies which it feels would be of interest and that the International Committee cooperate with the Section on Cardiovascular Surgery in carrying out the studies.

WHEREAS, the International Committee on Hypertension has now been established, and

WHEREAS, hypertension is one of the most important aspects of cardiovascular disease,

BE IT RESOLVED, that the International Committee on Hypertension make an international study on the use of drugs in the treatment of hypertension and that the results of this study be submitted to the Board of Regents of the College.

The closing Executive Session was held on September 11, at a luncheon given by the Pfizer-Taito Company at the Tokyo Kaikan. Dr. Seymour M. Farber, President-Elect, presided.

At this meeting the following officials presented the national flags of their countries for permanent display in the gallery of flags at the College headquarters in Chicago:

Austria	Anton Sattler, Vienna, Regent
Republic of China	Jung Chin Tao, Taipei, Taiwan, Governor
Colombia	Rafael J. Mejia, Medellin, Governor
Germany	Hugo W. Knipping, Cologne, Governor
Honduras	Ramon Larios, Tegucigalpa, Governor
Philippines	Miguel Canizares, Manila, Regent
Sweden	Erik Hedvall, Uppsala, Governor
Thailand	Thip Pholpoke, Bangkok, Governor
Turkey	Celal Ertug, Ankara, Governor

A total of 24 national flags, representing the College chapters in countries throughout the world, are on display in the Chicago headquarters. The custom was initiated by the Cuban Chapter at the annual meeting of the College in Chi-

Dr. Farber receiving the Turkish flag from Dr. Celal Ertug, Ankara, Governor, at the closing Executive Session.

cago, Illinois in 1956. It is anticipated that additional chapters will present their national flags at future international congresses of the College.

The following resolutions were adopted:

WHEREAS, standards for Fellowship in the American College of Chest Physicians in the United States and Canada have been maintained at a high level by requiring that all candidates for Fellowship hold board certification or that they successfully complete our oral and written examinations, and

WHEREAS, these standards must be maintained at a high level throughout the world,

BE IT RESOLVED, that all candidates for Fellowship in the College in all countries be required to complete Fellowship examinations unless a waiver of this requirement is granted by the Board of Regents, on request by the Regent or Governors of the candidate's country, and

BE IT FURTHER RESOLVED, that examinations for Fellowship be established at the earliest possible date in those countries in which this procedure has not already been adopted and that such examinations be conducted under the supervision of the Regent or Governor of the country or territory concerned or by his appointee.

WHEREAS, international committees on twenty-five aspects of chest disease have now been established, and

WHEREAS, it is the intention of the Board of Regents of the College that every country be represented on each of these committees, and

WHEREAS, these committees will play an important part in the activities of the College,

BE IT RESOLVED, that the Regents and Governors in all countries take immediate steps to prepare a list of their members whom they wish to recommend to the President of the College for appointment to the committees, and

BE IT FURTHER RESOLVED, that the international committees hold meetings every two years at the time and place of the international congresses on diseases of the chest sponsored by the Council on International Affairs of the American College of Chest Physicians and that in the interim the chairmen of the various committees carry on the committee activities by correspondence.

WHEREAS, the American College of Chest Physicians is an international society having 6403 members in 89 countries and territories throughout the world, and

WHEREAS, the annual Essay Contest sponsored by the College is an integral part of the program of encouraging the interest of medical students in all countries to conduct research in diseases of the chest,

BE IT RESOLVED, that all members of the College, and in particular the Regents and Governors throughout the world, encourage medical students to participate in the Essay Contest on Diseases of the chest.

BE IT RESOLVED, that the Board of Regents and Board of Governors of the American College of Chest Physicians do hereby extend their sincere gratitude to the following persons and groups for their splendid hospitality and cooperation in making the Fifth International Congress on Diseases of the Chest a great success:

Prime Minister Nobusuke Kishi and the cooperating Ministers
Prof. Taizo Kumagai, President of the congress
Prof. Yoneji Miyagawa, Vice President of the congress
Dr. Jo Ono and his staff, particularly Mrs. Tamura and Mrs. Hooker
Mr. Yano, President of the Daiichi Insurance Company
The members of the Japan Chapter of the College
The Imperial Hotel
Hankyu Air & Sea Service

Remarks were made by Prof. Miyagawa and Dr. Jo Ono, expressing the pleasure of the members of the Japan Chapter in having had the opportunity to meet College members from many parts of the world and especially in having

the honor of being host to the Fifth International Congress on Diseases of the Chest. Dr. Farber extended the appreciation of the College to the Japan Chapter for the organization of a brilliant congress and for the warm hospitality extended by the people of Japan.

The Executive Director, Mr. Murray Kornfeld, announced that at a meeting of the Executive Council of the College held in Tokyo on Wednesday, September 10, invitations for the Sixth International Congress on Diseases of the Chest to be held in 1960 were studied. He reported that it was unanimously agreed to accept the invitation extended by the Government of Austria to hold the 1960 international congress in Vienna. Dr. Anton Sattler, Regent of the College for Austria, who extended the invitation in behalf of his government, expressed his appreciation in the selection of Vienna and pledged the support of the medical associations of Austria in the organization of the congress.

The Committee on Nominations, under the chairmanship of Dr. Burgess L. Gordon, USA, presented the following slate of officers for election, which was unanimously adopted. Dr. Jo Ono, Japan, and Dr. Helge B. Wulff, Sweden, served as members of the committee.

REGENTS

Honorary Regents

Canada	William E. Ogden	Toronto
Greece	Nicolas Oeconomopoulos	Athens
Italy	Eugenio Morelli	Rome

Regents

Argentina	Gumersindo Sayago	Cordoba
Australia	Sir Harry Wunderly	Canberra
Austria	Anton Sattler	Vienna
Belgium	Lucien Brull	Liege
Southern Brazil	Manoel de Abreu	Rio de Janeiro
Northern Brazil	Jose Silveira	Salvador
Chile	Hector Orrego Puelma	Santiago
China, Republic of	Li Shu-Fan	Hong Kong
Colombia	Carlos Arboleda Diaz	Bogota
Cuba	Antonio Navarrete	Havana
Ecuador	Juan Tanca Marengo	Guayaquil
England	Richard R. Trail	London
France	Etienne Bernard	Paris
Germany	Joachim Hein	Innien
Greece	Basil N. Papanicolaou	Athens
India	Raman Viswanathan	New Delhi
Italy	A. Omodei Zorini	Rome
Japan	Jo Ono	Tokyo
Lebanon	Papken S. Mugrditchian	Beirut
Mexico	Donato G. Alarcon	Mexico City
Netherlands	Leendert D. Eerland	Groningen
Panama	Maximo Carrizo Villarreal	Colon
Peru	Ovidio Garcia-Rosell	Lima
Philippine Islands	Miguel Canizares	Manila
Portugal	Lopo de Carvalho	Lisbon
South Africa	David P. Marais	Cape Town
Spain	Antonio Caralps	Barcelona
Sweden	Clarence Crafoord	Stockholm
Switzerland	Wilhelm Loeffler	Zurich
Turkey	Tevfik Saglam	Istanbul
Uruguay	Fernando D. Gomez	Montevideo
Venezuela	Jose Ignacio Baldo	Caracas

GOVERNORS

Honorary Governors

Brazil	Reginaldo Fernandes	Rio de Janeiro
Italy	Maurizio Ascoli	Palermo

Governors

Argentina	Raul F. Vaccarezza	Buenos Aires
Australia		
New South Wales	Marshall Andrew	Sydney
Victoria	David B. Rosenthal	Melbourne
Austria	Erhard F. Kux	Innsbruck
Belgium	Henry Durieu	Brussels
Brazil		
Bahia	Manoel Ezequiel da Costa	Salvador
Minas Gerais	Orlando Cabral Motta	Belo Horizonte
Para	Epilogo de Campos	Belem
Pernambuco	Herodoto Pinheiro Ramos	Recife
Rio de Janeiro	Mauricio Teichholz	Rio de Janeiro
State of Rio	Antonio Jorge Abunahman	Niteroi
Rio Grande do Sul	Carlos Bento	Porto Alegre
Sao Paulo	E. de Jesus Zerbini	Sao Paulo
Vitoria	Jayme Santos Neves	Espirito Santo
Canada		
British Columbia	W. Elliott Harrison	Vancouver
Eastern Provinces	J. J. Quinlan	Kentville, N.S.
Manitoba	Lawrence R. Coke	Winnipeg
Ontario	John A. Lewis	Toronto
Quebec	B. Guy Begin	Montreal
Western Provinces	Leslie McR. Mullen	Calgary, Alberta
Ceylon	George E. Ranawake	Ratmalana
Chile		
Concepcion	Ildefonso Garreton Unda	Concepcion
Santiago	Armando Alonso Vial	Santiago
Valparaiso	Gilberto V. Zamorano	Valparaiso
China, Republic of	Jung Chin Tao	Taipei, Taiwan
Colombia	Rafael J. Mejia	Medellin
Costa Rica	Raul Blanco Cervantes	San Jose
Cuba	Francisco J. Menendez	Havana
Czechoslovakia	Jaroslav Jedlicka	Prague
Denmark	Jens L. Hansen	Copenhagen
Ecuador	Jorge A. Higgins	Guayaquil
Egypt	Abdel-Aziz Sami	Cairo
El Salvador	Jose Francisco Valiente	San Salvador
England		
Greater London	Geoffrey Bourne	London
Northern England	Peter W. Edwards	Shropshire
Southern England	Sir Geoffrey Todd	Midhurst
Finland	Sakari Mustakallio	Helsinki
France		
Lyon	Paul Santy	Lyon
Nantes	Paul Veran	Nantes
Paris	Maurice Bariety	Paris
Paris	Andre Meyer	Paris
Germany		
Cologne	H. W. Knipping	Cologne
Freiburg	Ludwig Heilmeyer	Freiburg
Gottingen	Rudolf Schoen	Gottingen
Hamburg	Josef Jacobi	Hamburg
Munich	Rudolf Zenker	Munich
West Berlin	Walter Unverricht	West Berlin
Wiesbaden	Hans Wurm	Wiesbaden
Wurzburg	Ernst Wollheim	Wurzburg

Governors (continued)

Greece	Panayiotis Chortis	Athens
Haiti	Louis Roy	Port-au-Prince
Honduras	Ramon Larios	Tegucigalpa
Hong Kong	Kenneth Hui	Hong Kong
India		
Eastern India	P. K. Ghosh	Calcutta
Northern India	K. L. Wig	New Delhi
Southern India	K. S. Sanjivi	Madras
Western India	B. R. Billimoria	Bombay
Iraq	Najib Mahmoud	Baghdad
Ireland	Victor M. Synge	Dublin
Israel	Juda M. Pauzner	Tel Aviv
Italy		
Milan	Giuseppe Daddi	Milan
Naples	Vincenzo Monaldi	Naples
Palermo	Nicola Sanguigno	Palermo
Rome	Giovanni L'Eltore	Rome
Japan		
Northeast Japan	Takashi Nakamura	Sendai
Southwest Japan	Imazoto Donomae	Osaka
Tokyo	Ichiro Akakura	Tokyo
Tokyo	Osami Kitamoto	Tokyo
Jordan	Rafat A. Faris	Amman
Korea	Eung Soo Han	Seoul
Lebanon	Elias Khoury	Beirut
Mexico	Miguel Jimenez Sanchez	Mexico
Netherlands	M. R. Heynsius van den Berg	Amsterdam
Nicaragua	Rene Vargas	Managua
Norway	Einar Murstad	Lillehammer
Pakistan, East	Mohammed Ibrahim	Dacca
Pakistan, West	Rocinton B. F. Khambatta	Karachi
Panama	Rodolfo V. Young	Ancon
Paraguay	Juan Max Boettner	Asuncion
Peru	Maximo Espinoza Galarza	Lima
Philippine Islands	Manuel Quisumbing, Sr.	San Pablo
Portugal	Carlos Alberto Vidal	Lisbon
South Africa		
Northern States	Maurice A. Pringle	Transvaal
Southern States	Theodore Schrire	Cape Town
Spain		
Andalusia	Carlos G. Zurita	Cabra
Barcelona	Raimundo Frouchtman	Barcelona
Bilbao	Carmelo Gil-Turner	Bilbao
Canary Islands	Tomas Cervia	Tenerife
La Coruna	Alvaro Urgoiti	La Coruna
Madrid	Jose Abello	Madrid
Sweden		
Gothenburg	Gosta Birath	Gothenburg
Malmo	Helge B. Wulff	Malmo
Uppsala	Erik Hedvall	Uppsala
Switzerland		
Central Switzerland	Alfred Brunner	Zurich
West Switzerland	Maurice Gilbert	Geneva
Syria	Bechir Azme	Damascus
Thailand	Thip Pholpoke	Bangkok
Turkey	Celal Ertug	Ankara
Uruguay	Armando Sarno	Montevideo
Venezuela		
Guarico Province	Julio Criollo Rivas	Caracas
Zulia Province	Pedro Iturbe	Maracaibo
Yugoslavia	Robert T. Neubauer	Sezana

Congress Banquet

The Congress Banquet, preceded by a cocktail party, was held on Wednesday night, September 10, at the Toyko Kaikan. Prof. Kumagai, President of the congress, presided on this gala occasion and gave a brief talk. A Torii with an engraved plaque, commemorating 25 years of College service, was presented to Mr. Murray Kornfeld by Prof. Kumagai in behalf of the Japan Chapter of the College. Entertainment was presented after the banquet by the world-famous Takarazuka performers who gave several song and dance numbers, including some lovely Japanese Folk Songs. Their performance was presented by courtesy of the Keihanshin Kyuko Railway Co., Ltd. The remainder of the evening was given over to dancing for which two orchestras alternated in providing continuous music.

Scientific Sessions

Two halls were used in the Daiichi Building for the presentation of the scientific program from 9 a.m. to 5 p.m., September 8, 9 and 10, and from 9 a.m. to 12 noon on September 11. On the afternoon of September 11, 27 subjects were presented at the fireside conferences held at the Tokyo Kaikan. All of these scientific sessions were filled to capacity.

The following panels were held in Hall No. 1: Tuberculosis; Cardiopulmonary Function; Coronary Disease; Benign and Malignant Tumors of the Chest; Aviation Medicine. In addition, there were formal lecture sessions on: Tuberculosis; Fungus Infections; Heart and Circulation; Emphysema; Benign and Malignant Tumors of the Chest; Cardiopulmonary Disease; Chest Disease in Pediatrics; Cardiovascular and Pulmonary Surgery; Occupational Diseases of the Chest; Bronchoesophagology; and Miscellaneous Topics. The scientific sessions were translated simultaneously, with the use of earphones, into the official languages of the congress, Japanese, French and English.

Motion picture sessions were presented during each day of the congress covering recent advances in the medical and surgical treatment of chest diseases. Films were also shown of cultural and industrial aspects of Japan. The film sessions were presented at the Red Cross Building, which also housed the technical and scientific exhibits of the congress, representing the leading Japanese pharmaceutical and medical supply houses.

The British Delegation, from left to right: Dr. David Rosenthal, Melbourne, Australia, Governor; Dr. Jethro Gough, Cardiff, Wales; Mrs. Crouch, daughter of Dr. Peter W. Edwards, Shropshire, England, Governor; Dr. E. A. McMaster, Seaforth, Ontario, Canada; and Dr. A. S. R. Peffers, London, England.

Congress Tour group arriving in Honolulu en route to Tokyo for the Fifth International Congress.

Social Events

The Hon. Nobusuke Kishi, Prime Minister of Japan, and Mrs. Kishi, gave a delightful reception in the beautiful garden of their official residence for the delegates of the Congress on Monday evening, September 8. The Prime Minister and his lady were gracious hosts and received their guests with great warmth and hospitality.

On Tuesday evening, September 9, twenty-six Embassies and Consulates in Tokyo gave receptions for the delegates from their respective countries. We quote from a letter received from Douglas MacArthur II, American Ambassador to Japan:

> "I have seldom met with a more distinguished group, nor a more pleasant one than the delegates to the Fifth International Congress of Chest Physicians. Such a successful meeting of distinguished professional men is indeed a beacon of what can be accomplished with good will and ability."

Delegates to the congress were received at a beautiful supper party at Chinzanso on the closing day, September 11, given by the Hon. Seiichiro Yasui, Governor of Tokyo. The supper buffet included such delicacies as never before tasted by our members from many parts of the world. It included Sukiyaki, Tempura, Ghengis Khan and many other dishes. The magnificent gardens of Chinzanso provided an incomparable atmosphere and background.

The Regents and Governors of the College were entertained by Prof. Taizo Kumagai, President of the congress, on Saturday evening, September 6, at a reception in the Imperial Hotel, and on Sunday they were taken to Fuchu to attend a garden party at the lovely villa of Mr. Shojiro Ishibashi, President of the Bridgestone Tire Company of Japan.

Administrative Sessions

On Saturday afternoon, September 6, the following international committees held meetings at the Tokyo Kaikan. This was the first meeting of the newly organized international committees and the interest and enthusiasm shown in their activities is most gratifying. The international committees will meet biennially at the international congresses of the College, and in the interim, their activities will be conducted through correspondence. Many of the committees have submitted reports covering their programs planned for the com-

The ladies kimono fashion show, Takashimaya Department Store, Tokyo.

Delegates attending the International Conference on Diseases of the Chest sponsored by the Hong Kong and China Chapter of the College. This photograph was taken at the Li Shu-Fan Block of the Hong Kong Sanatorium and Hospital, where the scientific program was presented.

ing two years. It is anticipated that additional members will be appointed by the President to each committee in order that countries throughout the world will be represented. College members who are interested in serving on these committees are invited to write to the President of the College for consideration of appointment.

International Committees

Undergraduate Medical Education	Psychosomatic Aspects of Chest Diseases
Postgraduate Medical Education	Chest Roentgenology
Chemotherapy and Antibiotics	BCG
Nonsurgical and Drug Therapy	Tuberculin Testing
Pulmonary Surgery	Rehabilitation in Pulmonary Disease
Bronchoesophagology	Clinical Cardiovascular Disease
Physiologic Therapy	Electrocardiography
Microbiology	Cardiovascular Physiology
Pulmonary Diseases in Children	Hypertension
Allergy	Angiocardiography
Occupational Diseases of the Chest	Pediatric Cardiology
Chest Diseases in Institutions	Cardiovascular Surgery

Rehabilitation in Cardiovascular Disease

The Editorial Board for *Diseases of the Chest*, the official journal of the American College of Chest Physicians, met at lunch on Saturday, September 6, at the Tokyo Kaikan. Dr. J. Arthur Myers, USA, Editor-in-Chief, presided at the meeting which was attended by the following members of the Editorial Board:

Donato G. Alarcon, Mexico	Edgar Mayer, USA
Jose Ignacio Baldo, Venezuela	Jo Ono, Japan
Andrew L. Banyai, USA	Coleman B. Rabin, USA
Miguel Canizares, Philippines	Henry C. Sweany, USA
Seymour M. Farber, USA	Raul F. Vaccarezza, Argentina
Ovidio Garcia-Rosell, Peru	Raman Viswanathan, India
Paul H. Holinger, USA	Attilio Omodei Zorini, Italy

Murray Kornfeld, Managing Editor, USA

Many matters relating to the publication of the College journal were discussed at this meeting.

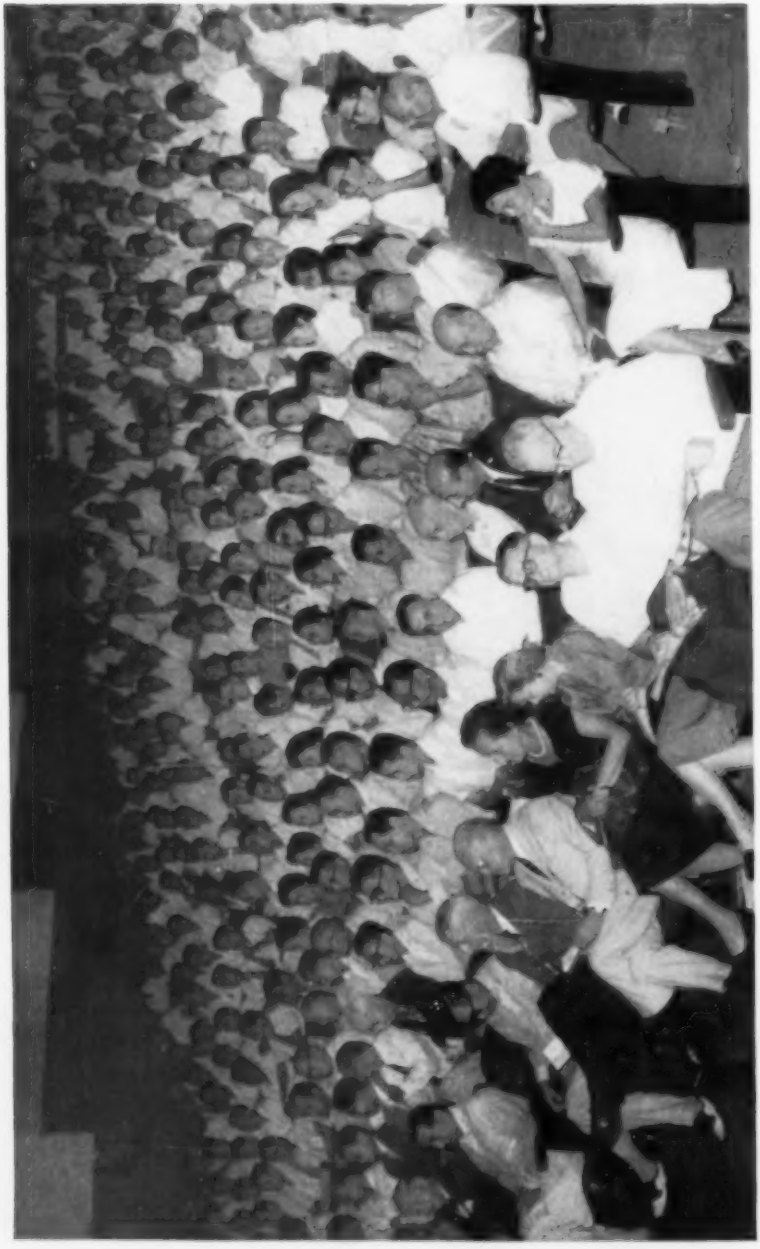
A luncheon meeting of the Executive Council of the College was held at the Imperial Hotel on Wednesday, September 10. The meeting was presided over by Dr. Seymour M. Farber, USA, President-Elect. Invitations were received from Austria, India, Turkey and the United States of America to be host for the Sixth International Congress on Diseases of the Chest in 1960. After serious deliberation it was unanimously voted to accept the invitation received from the Austrian Government through the Regent of the College for that country, Dr. Anton Sattler of Vienna.

Registration

The total registration for the congress was 1268, including physicians and their families, guests and exhibitors representing 35 countries and territories throughout the world. The countries and number of persons represented is shown in the following tabulation:

Argentina	5	China, Republic of	15	Pakistan	1
Australia	4	Colombia	3	Panama	1
Austria	1	England	4	Peru	4
Brazil	1	France	4	Philippines	31
Canada	4	Germany	7	Portugal	2
Ceylon	2	Greece	1	Sweden	8
Korea	12	Honduras	2	Thailand	13
Macau	1	Hong Kong	4	Turkey	1
Malaya	1	India	5	Venezuela	6
Mexico	5	Iran	2	Vietnam	1
Morocco	3	Italy	8	U.S.A.	184
Nicaragua	1	Japan	921		

35 countries—total registration 1268



The scientific session sponsored by the Philippine Chapter at the University of Santo Thomas, Manila, September 23.

Chapter Meetings

Hawaiian Chapter

A group of approximately eighty physicians and their families arrived in Honolulu from San Francisco on Friday, August 29, en route to the Fifth International Congress on Diseases of the Chest in Tokyo. On Saturday morning, August 30, the Hawaiian Chapter of the College sponsored a scientific program at the Mabel Smyth Auditorium, which was attended by more than one hundred physicians. Dr. Hastings H. Walker, Regent of the College for Hawaii, was in charge of arrangements.

Hong Kong and China Chapter

Following the International Congress in Tokyo, a group of one hundred physicians and their families visited Hong Kong, where the Hong Kong and China Chapter of the College sponsored an International Conference on Diseases of the Chest, September 17-21. Dr. Li Shu-Fan, Regent of the College, was chairman of arrangements for the conference, and Sister Mary Aquinas, President of the Chapter, assisted. The scientific program and fireside conferences were presented at the Hong Kong Sanatorium and Hospital. There was a splendid social program arranged for the visiting delegates and their families. American Consul-General J. B. Pilcher and Mrs. Pilcher gave a reception on September 18 in honor of His Excellency, the Governor of Hong Kong. A banquet was given on September 20 at the Cafe de Chine, Hong Kong, and Dr. Li entertained the group at garden parties given at his city residence, "White Jade," and his country residence, "Green Jade." There were visits to the Hong Kong Sanatorium and Hospital, the Ruttonjee Sanatorium and Grantham Hospital, as well as tours of Hong Kong, Kowloon and the New Territories.

Philippine Chapter

A meeting sponsored by the Philippine Chapter of the College was held in Manila during September 23-25, for the group arriving from Hong Kong. An excellent scientific program was presented at the auditorium of the University of Santo Tomas on September 23, which was attended by approximately one thousand physicians, residents, interns and medical students. The Philippine Chapter gave a luncheon and a banquet for the delegates, and the Philippine Tuberculosis Society sponsored a luncheon for the group. A courtesy call was paid the President of the Philippines, the Hon. Carlos P. Garcia, and the First Lady, at Malacanang Palace on September 25. A tour was made of the hospitals in Manila, including the Quezon Institute, the largest tuberculosis hospital in the Philippines of which Dr. Miguel Canizares, Regent of the College for the Philippines, is medical director. The Philippine Chapter officers, Dr. Jose R. Celis, President, Dr. A. S. Gaerlan, Vice President, Dr. Priscilla J. Tablan, Secretary-Treasurer, and Dr. Manuel Quisumbing Sr., Governor, are commended for the organization of an outstanding meeting.

Seventh International Congress of Bronchoesophagology

A group of more than one hundred physicians and their families traveled from Tokyo to Kyoto on September 12 to attend the Seventh International Congress of Bronchoesophagology. The Congress was officially opened at a reception given by the President, Professor Mitsuharu Goto. Excellent scientific programs were presented at the Kyoto University Hall on September 13 and 14. A congress banquet was given at the Miyako Hotel on September 13, and luncheons were given by the governor and mayor of Kyoto, and the governor and mayor of Nara.

SCIENTIFIC PROGRAM COMMITTEE REQUESTS ABSTRACTS FOR SILVER ANNIVERSARY MEETING

The 25th Annual Meeting of the College will be held at the Ambassador Hotel, Atlantic City, New Jersey, June 3-7, 1959. Special plans for the scientific program to be presented at the Silver Anniversary Meeting are now under way. Physicians who wish to present papers are urged to submit a 200-word abstract to the appropriate committee chairman by December 31, 1958 for consideration. Please forward abstracts to one of the following co-chairmen:

Dr. Arthur M. Master, 125 East 72nd Street, New York City
Chairman, Section on Cardiovascular Diseases

Dr. Coleman B. Rabin, 110 East End Avenue, New York City
Chairman, Section on Pulmonary Diseases

The Committee on Motion Pictures of the College will be interested to learn of new films on diseases of the chest for possible presentation at the 25th Annual Meeting in Atlantic City. All pertinent information concerning films should be forwarded to Dr. Paul H. Holinger, chairman of the committee, 112 East Chestnut Street, Chicago 11, Illinois. The committee will be pleased to review films for official approval and inclusion in the Approved Film List of the American College of Chest Physicians.

TRANS-ATLANTIC CONFERENCE SET ON CHEST DISEASE

American and British physicians attending medical meetings in their respective countries were linked together for a trans-Atlantic conference on diseases of the chest, December 5.

The American doctors were attending the 12th clinical meeting of the American Medical Association in Minneapolis, and the English doctors were attending a similar meeting of the British Medical Association in Southampton. They were linked via the underseas telephone cable.

The hour-long conference on lung and heart disease was presented in cooperation with Smith, Kline and French Laboratories, Philadelphia pharmaceutical house.

Physicians attending the A.M.A. meeting were able to hear the conference over a special high-fidelity system.

The American panel consisted of Dr. James R. Fox, Minneapolis chest physician, moderator; Dr. John F. Briggs, St. Paul specialist in internal medicine; and Dr. James A. Wier, a cardiopulmonary physiologist at the Army Chest center, Fitzsimons Army Hospital, Denver.

The English panel members were Dr. W. D. Brinton, Winchester, moderator; Dr. J. G. Scadding, a chest physician; Dr. T. Holmes Sellors, a thoracic surgeon; and Dr. J. McMicheal, a general physician, all of London.

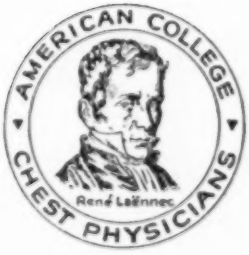
NEWS NOTE

Mr. H. B. Burns, President and Chairman of the Board of the U. S. Vitamin Corporation, and **Mr. I. A. Botty**, Vice President, accompanied **Dr. Hector Orrego Puelma**, Sanitago, Chile, Regent of the College for Chile, on an inspection of the laboratories in Santiago where **Dr. Casimir Funk**, internationally famous biochemist, is conducting cancer research. Dr. Orrego praised the Pan-American medical collaboration jointly effected by Arlington-Funk Laboratories Division of the U.S. Vitamin Corporation U.S.A., and Arlington-Funk Laboratories de Chile, Inc. These two companies confer an annual scholarship making it possible for a Chilean physician to visit university hospitals and laboratories in the United States. In 1958, the prize was given to Dr. Orrego, who is head of the Medical Department at the Hospital del Torax of Santiago and Director of the Medical Graduate School of the University of Chile.

DISEASES

of the

CHEST



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JULY-DECEMBER, 1958

DISEASES *of the* CHEST

OFFICIAL PUBLICATION
OF THE
AMERICAN COLLEGE OF CHEST PHYSICIANS

EXECUTIVE OFFICE
112 East Chestnut Street, Chicago 11, Illinois, U.S.A.
MURRAY KORNFELD, *Managing Editor*
WARD BENTLEY, *Business Manager*

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MEDICAL SERVICE BUREAU

POSITION WANTED

English-trained thoracic surgeon seeks appointment with hospital or group. Extensive experience in pulmonary, esophageal, cardiovascular and experimental surgery, teaching, chest medicine and administration. Please address inquiries to Box 302B, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

POSITIONS AVAILABLE

Physician wanted for 200-bed chronic disease and tuberculosis hospital. Large out-patient department. Attractive location. Starting salary $9,000 per year and up depending on qualifications. Part maintenance. Must be eligible for Michigan license. Give complete outline of education and professional experience with first letter. Please address inquiries to Box 299A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Physician wanted interested in pulmonary diseases for 170-bed state supported tuberculosis hospital established 3 years. Active surgical program, out-patient service, x-ray and laboratory. Consultants in all branches. U.S. citizenship and Alabama medical license or eligibility therefore required. Salary $8500 to $9500 depending on qualifications. Quarters to be made available. Apply: Medical Director, Sixth District Tuberculosis Hospital, 800 St. Anthony Street, Mobile, Alabama.

Residents wanted in pulmonary diseases for July 1, 1959 in 600-bed hospital in the Ozarks. Fully certified, excellent facilities, research and teaching programs, generous salary. Write: Charles A. Brasher, M.D., Superintendent and Medical Director, Missouri State Sanatorium, Mt. Vernon, Missouri.

CALENDAR OF EVENTS

NATIONAL MEETINGS

Twenty-fifth Annual Meeting
Silver Anniversary, American College of Chest Physicians
Ambassador Hotel, Atlantic City, New Jersey
June 3-7, 1959

Homecoming Meeting
Silver Anniversary, American College of Chest Physicians
Albuquerque, New Mexico, October 14-17, 1959

POSTGRADUATE COURSES

4th Annual Course on Diseases of the Chest
San Francisco, California, February 16-20, 1959

4th Regional Course on Diseases of the Chest
Newark, New Jersey, April 1, 8, 15, 22, 1959

12th Annual Course on Diseases of the Chest
Philadelphia, March 30-April 3, 1959

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monilial complications

---

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for protection against monilial complications

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